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=> s fertility (3a) treat?
L1 2613 FERTILITY (3A) TREAT?

=> s contracept?
L2 78848 CONTRACEPT?

=> s l1 or l2
L3 81300 L1 OR L2

=> s endometrium (3a) matur?
L4 142 ENDOMETRIUM (3A) MATUR?

=> s l3 or l4
L5 81423 L3 OR L4

=> s l4 and l3
L6 19 L4 AND L3

=> s progest? (3a) receptor?
L7 29173 PROGEST? (3A) RECEPTOR?

=> s (antagonist or inhibit?) (3a) l7
L8 1077 (ANTAGONIST OR INHIBIT?) (3A) L7

=> d bib abs

L8 ANSWER 1 OF 1077 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:325545 BIOSIS
DN PREV200200325545
TI The histone deacetylase ***inhibitor*** trichostatin A blocks ***progesterone*** ***receptor*** -mediated transactivation of the mouse mammary tumor virus promoter in vivo.
AU Wilson, Melissa A.; Ricci, Andrea r.; Deroo, Bonnie J.; Archer, Trevor K. (1)
CS (1) Chromatin and Gene Expression Section, Lab. of Reproductive and Developmental Toxicology, NIEHS, NIH, 111 Alexander Dr., MD E4-06, Research Triangle Park, NC, 27709: archer1@niehs.nih.gov USA
SO Journal of Biological Chemistry, (April 26, 2002) Vol. 277, No. 17, pp. 15171-15181. http://www.jbc.org/. print.
ISSN: 0021-9258.
DT Article
LA English
AB Post-translational modifications of histones play an important role in modulating gene transcription within chromatin. We used the mouse mammary tumor virus (MMTV) promoter, which adopts an ordered nucleosomal structure, to investigate the impact of a specific inhibitor of histone deacetylase, trichostatin A (TSA), on progesterone receptor-activated transcription. TSA induced global histone hyperacetylation, and this effect occurred independently of the presence of hormone. Interestingly, chromatin immunoprecipitation analysis revealed no significant change in the level of acetylated histones associated with the MMTV promoter following high TSA treatment. In human breast cancer cells, in which the MMTV promoter adopts a constitutively "open" chromatin structure, treatment with TSA converted the MMTV promoter into a closed structure. Addition of hormone did not overcome this TSA-induced closure of the promoter chromatin. Furthermore, TSA treatment resulted in the eviction of the transcription factor nuclear factor-1 from the promoter and reduced progesterone receptor-induced transcription. Kinetic experiments revealed that a loss of chromatin-remodeling proteins was coincident with the decrease in MMTV transcriptional activity and the imposition of repressed chromatin architecture at the promoter. These results demonstrate that deacetylase inhibitor treatment at levels that induce global histone acetylation may leave specific regulatory regions relatively unaffected and that this treatment may lead to transcriptional inhibition by mechanisms that modify chromatin-remodeling proteins rather than by influencing histone acetylation of the local promoter chromatin structure.

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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 11:19:22 ON 06 JUN 2002
L1 2613 S FERTILITY (3A) TREAT?
L2 78848 S CONTRACEPT?
L3 81300 S L1 OR L2
L4 142 S ENDOMETRIUM (3A) MATUR?
L5 81423 S L3 OR L4
L6 19 S L4 AND L3
L7 29173 S PROGEST? (3A) RECEPTOR?
L8 1077 S (ANTAGONIST OR INHIBIT?) (3A) L7

=> s 17 alpha fluoralkyl?
L9 0 17 ALPHA FLUORALKYL?

=> s 17 fluoralky?
L10 0 17 FLUORALKY?

=> s 17 (3a) fluro?
L11 8 17 (3A) FLURO?

=> d bib abs

L11 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997:83613 BIOSIS
DN PREV199799375326
TI Radiofrequency ablation in multiple accessory pathways.
AU Iturralde Torres, Pedro; Lara, Susano; Picos Bovio, Eva; Colin Lizalde, Luis; Kershenovich, Sergio; Gonzalez Hermosillo, J. Antonio
CS Inst. Nac. Cardiol., Dep. Electrofisiol., Juan Badiano Number 1, 14080 Mexico, DF Mexico
SO Archivos del Instituto de Cardiologia de Mexico, (1996) Vol. 66, No. 5, pp. 390-399.

ISSN: 0365-3080.
DT Article
LA Spanish
SL Spanish; English; French
AB Radiofrequency ablation were performed in 465 consecutive patients with Wolff-Parkinson-White syndrome. We demonstrated evidence of multiple accessory pathways in 18 patients (3.9%); 17 had two, and one had three accessory pathways. In three of these there was additional right atrio-fascicular connection. There were 27 manifest and 10 concealed accessory pathways. Six patients had Ebstein's anomaly associated with accessory pathways. The location of the accessory pathways was in the right posterior portion of the ventricular septum in 16, the left posterior portion of the ventricular septum in 5, the right free wall in 5, the left free wall in 7, the right anteroseptal in 3, the midseptum in 1, and one right anterolateral. Of the 37 pathways, 32 (86%) accessory pathways were ablated successfully without complications. Duration of the procedure was 100+-58 minutes, and ***fluroscopic*** time 40+-***17*** minutes. The mean applications were 26+-17 and the power level of the radiofrequency current 41+-6 W. A follow-up of 80+-40 days after ablation demonstrated incidence of recurrent conduction in 3 accessory pathways (8%). In conclusion, patients with multiple accessory pathways can be treated by radiofrequency ablation in a single session with a high success rate although slightly less than that inpatients with a single accessory pathway.

=> s 17 (3A) fluor?
L12 3684 17 (3A) FLUOR?

=> s l12 (3a) alkyl?
L13 15 L12 (3A) ALKYL?

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L8 1077 S (ANTAGONIST OR INHIBIT?) (3A) L7
L9 0 S 17 ALPHA FLUORALKYL?
L10 0 S 17 FLUORALKY?
L11 8 S 17 (3A) FLURO?
L12 3684 S 17 (3A) FLUOR?
L13 15 S L12 (3A) ALKYL?

=> s l13 and l8
L14 0 L13 AND L8

=>

=> s l8 and l3
L15 69 L8 AND L3

=> s l15 and l6
L16 1 L15 AND L6

=> d bib abs

L16 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
AN 2000:854126 CAPLUS
DN 134:37152
TI The use of progesterone antagonists and progesterone receptor modulators in ***contraception***
AU Spitz, I. M.; Van Look, P. F. A.; Coelingh Bennink, H. J. T.
CS Institute of Hormone Research, Shaare Zedek Medical Center, Jerusalem, Israel
SO Steroids (2000), 65(10-11), 817-823
CODEN: STEDAM; ISSN: 0039-128X
PB Elsevier Science Inc.
DT Journal; General Review
LA English
AB A review, with 59 refs. Progesterone antagonists (PAs) and progesterone receptor modulators (PRMs) have ***contraceptive*** potential by suppressing follicular development, delaying the surge of LH, retarding endometrial maturation, and promoting endometrial bleeding. Mifepristone, in daily doses of 2-10 mg, blocks the LH surge and ovulation. Many of the studies were conducted in women not at risk of pregnancy, and thus the ***contraceptive*** efficacy is not yet known. Nevertheless, there is evidence that daily doses of 2 or 5 mg of mifepristone have ***contraceptive*** potential. Because of anovulation, there may be an unopposed estrogen effect on the endometrium, although this risk may be mitigated by the noncompetitive anti-estrogenic activity exhibited by both PAs and PRMs. Low doses of PAs and PRMs, which do not affect ovulation, retard endometrial ***maturation*** , indicating that the ***endometrium*** is exquisitely sensitive to these compds. This raises the prospect of endometrial ***contraception*** , i.e. prevention of endometrial maturation without disturbing ovulation or producing alterations in bleeding patterns. This approach works well in monkeys but was not found to be very promising when given to women not using ***contraception*** . On the other hand, 200 mg mifepristone administered 48 h after the LH surge, which has minimal or no effect on ovulation and bleeding patterns, is an effective ***contraceptive*** ; yet, it is not a practical approach to ***contraception*** . Late luteal phase administration of mifepristone produces menstrual bleeding. However, when mifepristone was administered every month at the end of the cycle either alone or together with prostaglandins, it was not very effective in preventing pregnancy. In contrast, a mifepristone-prostaglandin combination has been shown to be a very effective treatment for occasional menstrual regulation, with vaginal bleeding induced in 98% of pregnant women, with menses delay of 11 days or less. Mifepristone is an excellent agent for emergency ***contraception*** when used within 120 h of unprotected intercourse. It is also possible that PAs and PRMs may be used to reduce the occurrence of bleeding irregularities induced by progestin-only ***contraceptive*** methods. Both classes of progesterone receptor ligands may also have ***contraceptive*** efficacy by having a pharmacol. effect on the embryo or altering tubal transport or other aspects of tubal physiol.
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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 11:19:22 ON 06 JUN 2002
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L9 0 S 17 ALPHA FLUORALKYL?
L10 0 S 17 FLUORALKY?
L11 8 S 17 (3A) FLURO?
L12 3684 S 17 (3A) FLUOR?
L13 15 S L12 (3A) ALKYL?
L14 0 S L13 AND L8
L15 69 S L8 AND L3
L16 1 S L15 AND L6

=> s conception?
L17 33312 CONCEPTION?

=> s l17 or l1
L18 35752 L17 OR L1

=> s l18 and l4 and l8
L19 0 L18 AND L4 AND L8

=> s l8 and l4
L20 1 L8 AND L4

=> d bib abs

L20 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
AN 2000:854126 CAPLUS
DN 134:37152
TI The use of progesterone antagonists and progesterone receptor modulators in contraception
AU Spitz, I. M.; Van Look, P. F. A.; Coelingh Bennink, H. J. T.
CS Institute of Hormone Research, Shaare Zedek Medical Center, Jerusalem, Israel
SO Steroids (2000), 65(10-11), 817-823
CODEN: STEDAM; ISSN: 0039-128X
PB Elsevier Science Inc.
DT Journal; General Review
LA English
AB A review, with 59 refs. Progesterone antagonists (PAs) and progesterone receptor modulators (PRMs) have contraceptive potential by suppressing follicular development, delaying the surge of LH, retarding endometrial maturation, and promoting endometrial bleeding. Mifepristone, in daily doses of 2-10 mg, blocks the LH surge and ovulation. Many of the studies were conducted in women not at risk of pregnancy, and thus the contraceptive efficacy is not yet known. Nevertheless, there is evidence that daily doses of 2 or 5 mg of mifepristone have contraceptive potential. Because of anovulation, there may be an unopposed estrogen effect on the endometrium, although this risk may be mitigated by the noncompetitive anti-estrogenic activity exhibited by both PAs and PRMs. Low doses of PAs and PRMs, which do not affect ovulation, retard endometrial ***maturation*** , indicating that the ***endometrium*** is exquisitely sensitive to these compds. This raises the prospect of endometrial contraception, i.e. prevention of endometrial maturation without disturbing ovulation or producing alterations in bleeding patterns. This approach works well in monkeys but was not found to be very promising when given to women not using contraception. On the other hand, 200 mg mifepristone administered 48 h after the LH surge, which has

minimal or no effect on ovulation and bleeding patterns, is an effective contraceptive; yet, it is not a practical approach to contraception. Late luteal phase administration of mifepristone produces menstrual bleeding. However, when mifepristone was administered every month at the end of the cycle either alone or together with prostaglandins, it was not very effective in preventing pregnancy. In contrast, a mifepristone-prostaglandin combination has been shown to be a very effective treatment for occasional menstrual regulation, with vaginal bleeding induced in 98% of pregnant women, with menses delay of 11 days or less. Mifepristone is an excellent agent for emergency contraception when used within 120 h of unprotected intercourse. It is also possible that PAs and PRMs may be used to reduce the occurrence of bleeding irregularities induced by progestin-only contraceptive methods. Both classes of progesterone receptor ligands may also have contraceptive efficacy by having a pharmacol. effect on the embryo or altering tubal transport or other aspects of tubal physiol.
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L12 3684 S 17 (3A) FLUOR?
L13 15 S L12 (3A) ALKYL?
L14 0 S L13 AND L8
L15 69 S L8 AND L3
L16 1 S L15 AND L6
L17 33312 S CONCEPTION?
L18 35752 S L17 OR L1
L19 0 S L18 AND L4 AND L8
L20 1 S L8 AND L4

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PROCESSING COMPLETED FOR L6
L21 8 DUP REM L6 (11 DUPLICATES REMOVED)

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L21 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2001:45304 BIOSIS
DN PREV200100045304
TI The use of progesterone antagonists and progesterone receptor modulators in ***contraception*** .
AU Spitz, Irving M. (1); Van Look, Paul F. A.; Coelingh Bennick, Herjan J. T.
CS (1) Institute of Hormone Research, Shaare Zedek Medical Center, Jerusalem: spitz@popcbr.rockefeller.edu Israel
SO Steroids, (October November, 2000) Vol. 65, No. 10-11, pp. 817-823. print. ISSN: 0039-128X.
DT Article
LA English
SL English
AB Progesterone antagonists (PAs) and progesterone receptor modulators (PRMs) have ***contraceptive*** potential by suppressing follicular development, delaying the surge of luteinizing hormone (LH), retarding endometrial maturation, and promoting endometrial bleeding. Mifepristone, in daily doses of 2-10 mg, blocks the LH surge and ovulation. Many of the studies were conducted in women not at risk of pregnancy, and thus the ***contraceptive*** efficacy is not yet known. Nevertheless, there is evidence that daily doses of 2 or 5 mg of mifepristone have ***contraceptive*** potential. Because of anovulation, there may be an unopposed estrogen effect on the endometrium, although this risk may be mitigated by the noncompetitive anti-estrogenic activity exhibited by both PAs and PRMs. Low doses of PAs and PRMs, which do not affect ovulation, retard endometrial ***maturation*** , indicating that the ***endometrium*** is exquisitely sensitive to these compounds. This raises the prospect of endometrial ***contraception*** , i.e. prevention of endometrial maturation without disturbing ovulation or producing alterations in bleeding patterns. This approach works well in monkeys but was not found to be very promising when given to women not using ***contraception*** . On the other hand, 200 mg mifepristone administered 48 h after the LH surge, which has minimal or no effect on ovulation and bleeding patterns, is an effective ***contraceptive*** ; yet, it is not a practical approach to ***contraception*** . Late luteal phase administration of mifepristone produces menstrual bleeding. However, when mifepristone was administered every month at the end of the cycle either alone or together with prostaglandins, it was not very effective in preventing pregnancy. In contrast, a mifepristone-prostaglandin combination has been shown to be a very effective treatment for occasional menstrual regulation, with vaginal bleeding induced in 98% of pregnant women, with menses delay of 11 days or less. Mifepristone is an excellent agent for emergency ***contraception*** when used within 120 h of unprotected intercourse. It is also possible that PAs and PRMs may be used to reduce the occurrence of bleeding irregularities induced by progestin-only ***contraceptive*** methods. Both classes of progesterone receptor ligands may also have ***contraceptive*** efficacy by having a pharmacological effect on the embryo or altering tubal transport or other aspects of tubal physiology.

L21 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
AN 1998:173048 BIOSIS
DN PREV199800173048
TI Vascular endothelial growth factor is essential for corpus luteum angiogenesis.
AU Ferrara, Napoleone (1); Chen, Helen; Davis-Smyth, Terri; Gerber, Hans-Peter; Thuy-Nhung Nguyen; Peers, David; Chisholm, Vanessa; Hillan, Kenneth J.; Schwall, Ralph H.
CS (1) Dep. Cardiovasc. Res., Process Sci. Pathol. Mol. Oncol., Genentech Inc., 1 DNA Way, San Francisco, CA 94080 USA
SO Nature Medicine, (March, 1998) Vol. 4, No. 3, pp. 336-340. ISSN: 1078-8956.
DT Article
LA English
AB The development and endocrine function of the ovarian corpus luteum (CL) are dependent on the growth of new capillary vessels. Although several molecules have been implicated as mediators of CL angiogenesis, at present there is no direct evidence for the involvement of any. Here we report the unexpected finding that treatment with truncated soluble Flt-1 receptors, which inhibit vascular endothelial growth factor (VEGF) bioactivity, resulted in virtually complete suppression of CL angiogenesis in a rat model of hormonally induced ovulation. This effect was associated with inhibition of CL development and progesterone release. Failure of ***maturation*** of the ***endometrium*** was also observed. Areas of ischemic necrosis were demonstrated in the corpora lutea (CLs) of treated animals. However, no effect on the preexisting ovarian vasculature was observed. These findings demonstrate that, in spite of the redundancy of potential mediators, VEGF is essential for CL angiogenesis. Furthermore, they have implications for the control of ***fertility*** and the ***treatment*** of ovarian disorders characterized by hypervascularity and hyperplasia.

L21 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
AN 1997:117292 BIOSIS
DN PREV199799416495
TI Midluteal immunoreactive alpha-inhibin serum concentrations as markers of luteal phase deficiency.
AU Balasch, Juan (1); Creus, Montserrat; Fabregues, Francisco; Casamitjana, Roser; Ordi, Jaime; Vanrell, Juan A.
CS (1) Dep. Obstet. Gynaecol., Fac. Med.-Univ. Barcelona, Barcelona Spain
SO Human Reproduction (Oxford), (1996) Vol. 11, No. 12, pp. 2591-2594. ISSN: 0268-1161.
DT Article
LA English
AB The present prospective clinical study was undertaken to determine the usefulness of midluteal phase serum immunoreactive alpha-inhibin concentrations as markers of luteal phase deficiency and whether they are better indicators of biopsy confirmed luteal phase defect than serum progesterone. Consecutive patients (n = 138) with regular menstrual cycles attending our Infertility Clinic (experimental group) and 5 fertile women who were requesting ***contraception*** and had regular menstrual patterns (control group) were included. In all women (patients and controls), basal body temperature, midluteal serum concentrations of oestradiol, prolactin, progesterone and immunoreactive alpha-inhibin, and premenstrual endometrial biopsy were used in the same cycle to assess luteal function. Out-of-phase secretory endometria were detected in 15 of the 138 patients. Thus, hormonal concentrations were compared between the following three groups of women: group 1 (n = 15), infertile patients with defective secretory endometria; group 2 (n = 123), infertile patients with normal secretory endometria; and controls (n = 15), fertile women with normal secretory endometria. Midluteal serum concentrations of progesterone, immunoreactive alpha-inhibin, oestradiol, and prolactin of the two groups studied were similar to those of the control group of fertile women. Our results indicate that midluteal serum inhibin determination does not accurately reflect histological ***maturation*** of the ***endometrium*** and it is not a better indicator of endometrial luteal phase deficiency than midluteal serum progesterone concentration.

L21 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 4
AN 1994:537392 BIOSIS
DN PREV199497550392
TI The effect of antiprogestin (RU 486) and prostaglandin biosynthesis inhibitor (naproxen) on uterine fluid prostaglandin F-2-alpha concentrations.
AU Gemzell-Danielsson, Kristina (1); Hamberg, Mats
CS (1) Dep. Obstet. Gynaecol., Karolinska Hosp., S-171 76 Stockholm Sweden

SO Human Reproduction (Oxford), (1994) Vol. 9, No. 9, pp. 1626-1630.
ISSN: 0268-1161.
DT Article
LA English
AB In the present study the effect of the antiprogestin RU 486 and the prostaglandin biosynthesis inhibitor, naproxen, on uterine fluid concentration of prostaglandin F-2alpha (PGF-2alpha) was investigated. RU 486, 200 mg, was administered two days after the luteinizing hormone (LH) surge and naproxen, 500 mg, was given every 12th hour five times starting 4 days after the LH surge. Uterine fluid was collected in the proliferative phase at ovulation and in the mid-luteal phase in a control and treatment cycle. The amount of PGF-2alpha was measured by gm chromatography-mass spectrometry. In the control cycle, the highest concentration of PGF-2alpha was found in the mid-luteal phase, and the lowest at the time of ovulation. Both RU 486 and naproxen reduced the PGF-2alpha concentration in uterine fluid considerably, or to 22-25% of that in the control cycle at the time of implantation. PGF-2alpha produced by the endometrium is believed to be of importance for the implantation of the blastocyst. Postovulatory treatment with RU 486 effectively prevents implantation, probably mainly by inhibiting the ***maturation*** of the ***endometrium*** during the secretory phase of the cycle. It is suggested that the inhibition of PGF-2alpha. release through the uterine fluid caused by RU 486 may also be of importance.

L21 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 5
AN 1989:430476 BIOSIS
DN BA88:88734
TI ***CONTRACEPTIVE*** POTENTIAL OF RU-486 BY OVULATION INHIBITION I.
PITUITARY VERSUS OVARIAN ACTION WITH BLOCKAGE OF ESTROGEN-INDUCED
ENDOMETRIAL PROLIFERATION.
AU VAN UEM J F H M; HSIU J G; CHILLIK C F; DANFORTH D R; ULMANN A; BAULIEU E
E; HODGEN G D
CS JONES INST. REPRODUCTIVE MED., DEP. OBSTET. GYNECOL., EASTERN VA. MED.
SCH., 855 WEST BRAMBLETON AVE., SUITE B, NORFOLK, VA. 23510.
SO CONTRACEPTION, (1989) 40 (2), 171-184.
CODEN: CCPTAY. ISSN: 0010-7824.
FS BA; OLD
LA English
AB In previous studies, RU 486 administration arrested spontaneous folliculogenesis. To investigate the central versus peripheral effects of RU 486 on the ovarian/menstrual cycle, including endometrial proliferation, RU 486 was administered daily (10 mg/kg/day, im) from menstrual cycle day 3 or 7 to day 25 in normal adult cynomolgus monkeys receiving hMG treatment (37.5 IU/day) from days 3-8 (n = 6). RU 486 administration with hMG/hCG therapy did not inhibit ovarian response, as evidenced by steroidogenesis and ovulation. Nine of 23 oocytes retrieved by lavage or follicular aspiration at laparotomy after ovulation induction were morphologically classified as mature preovulatory status. Whereas an endometrial biopsy performed on cycle day 25 in control monkeys revealed an in phase ***mature*** secretory ***endometrium*** , histologic sections from RU 486 plus hMG/hCG treated females uniformly demonstrated atrophic to weakly proliferative endometrium on cycle day 25, despite serum estradiol levels > 300 pg/ml. Three months after the initial 25-day study endometrial biopsies revealed persistent atrophic endometrium, even though repeated ovulation induction with hMG/hCG therapy elevated serum estrogen concentrations. The finding prevailed whether RU 486 treatment began on cycle day 3 or 7. The intermenstrual interval was significantly ($P < 0.01$) lengthened by RU 486 treatments (28.5 .+-. 2.0, control vs 131.3 .+-. 11.5 days, RU 486). In summary, RU 486 consistently blocked ovulation unless hCG was provided and elicited a persistent retardation of early proliferative endometrium when administered daily beginning in early or mid-follicular phase. The normal mitogenic effects of elevated ovarian estrogen secretion on endometrial tissue were quelled, uniformly resulting in amenorrhea. The long-lasting action of RU 486, causing ovulation inhibition and atrophic endometrium, may be due to the depot effect of im injection. In addition, RU 486 did not prevent ovarian steroidogenesis, ovulation or oocyte maturation when an ovulation induction regimen of hMG/cCG was given. These findings show that RU 486 prevented ovulation by diminishing pituitary gonadotropin secretion, rather than by direct effects on ovarian folliculogenesis, and induced amenorrhea by inhibiting estrogen-induced endometrial proliferation.

L21 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 6
AN 1988:74501 BIOSIS
DN BA85:40800
TI ENDOMETRIAL HISTOLOGY DURING INTERMITTENT INTRANASAL LHRH AGONIST
SEQUENTIALLY COMBINED WITH AN ORAL PROGESTOGEN AS AN ANTIOVULATORY
CONTRACEPTIVE APPROACH.
AU LEMAY A; JEAN C; FAURE N
CS CENTRE DE RECHERCHE ET DEP. DE GYNECOLOGIE-OBSTETRIQUE.
SO FERTIL STERIL, (1987) 48 (5), 775-782.
CODEN: FESTAS. ISSN: 0015-0282.
FS BA; OLD
LA English
AB Endometrial biopsies were performed in four groups of six or seven women treated for periods of 14 or 21 days with 200 .mu.g twice daily or 400 .mu.g once daily or intranasal Buserelin acetate. Five milligrams of medroxyprogesterone acetate (MPA) was taken orally twice daily on days 15 to 21. A medication-free week followed each treatment period. Between days 12 and 15 of the first treatment cycle, a proliferative endometrium was described in 16 out of 24 biopsies (66%). In 8 specimens (33%), early secretory changes were related to an early and/or short-lived rise in serum progesterone (P). At the end of the fourth treatment cycle, advanced maturation (days 23 to 28) was observed mainly in the 14-day schedules where serum estradiol (E2) was stimulated in or above the normal range of control cycles. Early to midluteal phase dating (days 16 to 22) was described mainly in the 21-day schedules. There was no P elevation in these groups. Five biopsies showing only proliferative tissue were associated with low levels of E2 mainly in the 400 .mu.g/day group. The regimen capable of maintaining E2 in the low physiologic range (200 .mu.g/12 hours .times. 21 days) was associated with incomplete secretory changes of the endometrium. A longer period of progestogen administration should produce a more complete ***maturation*** of the ***endometrium*** .

L21 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2002 ACS
AN 1976:504445 CAPLUS
DN 85:104445
TI Fructose metabolism in the rat endometrium under normal and experimental conditions
AU Boshier, D. P.; Katz, June M.
CS Dep. Anat., Univ. Auckland, Auckland, N. Z.
SO J. Reprod. Fertil. (1976), 47(2), 245-9
CODEN: JRPFA4
DT Journal
LA English
GI

/ Structure 1 in file .gra /

AB Fructose 1,6-diphosphate (FDP) [488-69-7] hydrolysis, which was measured in tissue exts. from ***mature*** rat ***endometrium*** , was maximal during late diestrus and early proestrus and reflected the variations in the plasma levels of ovarian steroids. Treatment of ovariectomized animals with estradiol benzoate (I benzoate) [50-50-0] or medroxyprogesterone acetate [71-58-9] increased FDP hydrolysis when compared with control animals, although I was the more effective. FDP hydrolysis was greater in exts. of endometrial tissue from uterine horns contg. a silk IUD or of deciduomal tissue from pseudopregnant rats, in which the peak was on day 9, than in exts. of endometrium from the control contralateral horn. The linking of glycogen metab. in normal endometrium, IUD-contg. horns, and decidual tissue to the role of FDP in gluconeogenesis and the promotion of glycogen storage is discussed.

L21 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
AN 1971:120826 CAPLUS
DN 74:120826
TI Activators of coagulation and fibrinolytic systems in normal and drug-influenced [menstrual] cycles
AU Stein, W. W.; Halberstadt, E.; Schwedes, R.
CS Univ.-Frauenklin., Frankfurt/M., Ger.
SO Fortschr. Med. (1971), 89(1), 17-20, 40
CODEN: FMDZAR
DT Journal
LA German
AB In addn. to thromboplastic activity, which showed no cyclic changes, the ***endometrium*** of sexually ***mature*** females was found to contain fibrinolytic activity, predominantly an activator which converts plasminogen into plasmin. The concn. of this activator increased in the menstrual secretion phase. Activator activity, but not thromboplastic activity, decreased during treatment with estrogen-gestagen prepns. Treatment with synthetic antifibrinolytics almost completely eliminated tissue activator activity.

=> d his

(FILE 'HOME' ENTERED AT 11:18:52 ON 06 JUN 2002)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 11:19:22 ON 06 JUN 2002
L1 2613 S FERTILITY (3A) TREAT?
L2 78848 S CONTRACEPT?
L3 81300 S L1 OR L2
L4 142 S ENDOMETRIUM (3A) MATUR?
L5 81423 S L3 OR L4
L6 19 S L4 AND L3
L7 29173 S PROGEST? (3A) RECEPTOR?
L8 1077 S (ANTAGONIST OR INHIBIT?) (3A) L7
L9 0 S 17 ALPHA FLUORALKYL?
L10 0 S 17 FLUORALKY?
L11 8 S 17 (3A) FLURO?
L12 3684 S 17 (3A) FLUOR?
L13 15 S L12 (3A) ALKYL?
L14 0 S L13 AND L8
L15 69 S L8 AND L3
L16 1 S L15 AND L6

L17 33312 S CONCEPTION?
L18 35752 S L17 OR L1
L19 0 S L18 AND L4 AND L8
L20 1 S L8 AND L4
L21 8 DUP REM L6 (11 DUPLICATES REMOVED)

=> dup rem l13
PROCESSING COMPLETED FOR L13
L22 15 DUP REM L13 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 15 ANSWERS - CONTINUE? Y/(N):y

L22 ANSWER 1 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 2000:750323 CAPLUS
DN 133:321996
TI Preparation of chlorosilanes
IN Chang, Il Nam; Yoo, Bok Yeul; Cho, Yeun Sok; Hahn, Joon soo
PA Korea Institute of Science and Tecnology, S. Korea
SO Jpn. Kokai Tokkyo Koho, 11 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2000297091	A2	20001024	JP 2000-15275	20000125
KR 2000066127	A	20001115	KR 1999-13006	19990413
PRAI KR 1999-13006	A	19990413		

OS CASREACT 133:321996; MARPAT 133:321996
AB R4CH2SiR1Cl2 (R1 = H, Cl, C1-6 ***alkyl*** , Ph; R4 = C1- ***17*** ***alkyl*** , C1-10 ***fluoroalkyl*** , C2-5 alkenyl, (CH2)nSiMe3-mClm, (substituted) arom. hydrocarbyl, (CH2)pSiR1Cl2, ArCH2SiR1Cl2; Ar = C6-14 arom. hydrocarbyl; n = 0-2; m = 0-3; p = 0-9) are prepd. by dehydrohalogenation of HSiR1Cl2 (R1 = same as above) with R2CH2X [R2 = C1- ***17*** ***alkyl*** , C1-10 ***fluoroalkyl*** , C2-5 alkenyl, (CH2)nSiMe3-mClm, (substituted) arom. hydrocarbyl, halo(alkyl), (halomethyl)aryl; X = Cl, Br; n, m = same as above] in the presence of tertiary phosphines. E.g., 1-chlorohexane was treated with HSiCl3 in the presence of Bu3P to give 65% hexyltrichlorosilane.

L22 ANSWER 2 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1998:558823 CAPLUS
DN 129:161760
TI Antigestagenically active steroids with ***fluorinated*** ***17*** .alpha.- ***alkyl*** chain
IN Schwede, Wolfgang; Cleve, Arwed; Klar, Ulrich; Neef, Guenter; Chwalisz, Kristof; Schneider, Martin; Fuhrmann, Ulrike; Hess-Stumpp, Holger
PA Schering A.-G., Germany
SO Ger. Offen., 10 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 19706061	A1	19980813	DE 1997-19706061	19970207
ZA 9800985	A	19990803	ZA 1998-985	19980206
WO 9834947	A1	19980813	WO 1998-EP752	19980209
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9861005	A1	19980826	AU 1998-61005	19980209
AU 742834	B2	20020110		
EP 970103	A1	20000112	EP 1998-905419	19980209
EP 970103	B1	20020417		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9807667	A	20000215	BR 1998-7667	19980209
JP 2001510479	T2	20010731	JP 1998-533785	19980209
NO 9903811	A	19991004	NO 1999-3811	19990806
US 6316432	B1	20011113	US 2000-516359	20000301
CN 1324802	A	20011205	CN 2000-129015	20000925
US 2002045774	A1	20020418	US 2001-978689	20011018
PRAI DE 1997-19706061	A	19970207		
US 1998-20947	B1	19980209		
WO 1998-EP752	W	19980209		
US 2000-516359	XX	20000301		

OS MARPAT 129:161760
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Title compds. I [R1 = Me, Et; R2 = CnFmHo; n = 2, 3, 4, 5, 6; m > 1; m+o = 2n+1; R3 = (un)etherized OH; R4, R5 = H, or R4R5 = bond, CH2; St = steroidal partial structure Q1-Q3; R6 = H, alkyl, halo; R7 = H, alkyl; or R6R7 = bond when St = Q1 or Q2; X = O, HO-N:, or (H,H); R8 = Y, aryl group (un)substituted by Y; Y = H, halo, OH, NO2, N3, cyano, substituted amino, acyl, etc.] are prepd. Thus, II was prepd. in 5 steps from 4-[3,3:17,17-bis(ethylenedioxy)estr-5-en-11.beta.-yl]phenol and perfluorononyl fluoride via condensation, deacetaliztion, addn. reaction with pentafluoroethyl iodide, reaction with (1-ethoxyethenyl)tributylstannane, and hydrolysis-isomerization. In an in vivo test, II at 0.1 mg/animal/day effected a 100% abortion rate in rats.

L22 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1991:138934 CAPLUS
DN 114:138934
TI Modification of endopeptidase for enhancing enzymic activity in organic solvents
IN Kawasaki, Norihiro; Takashita, Katsushige
PA Sanshin Chemical Industry Co., Ltd., Japan; Snow Brand Milk Products Co., Ltd.
SO Jpn. Kokai Tokkyo Koho, 7 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 02234674	A2	19900917	JP 1989-55053	19890309

OS MARPAT 114:138934
AB Endopeptidase is modified by introduction of RC(O) groups (e.g. benzyloxy, tert-butoxy, 9- ***fluorenylmethoxy*** , p-methoxybenzyloxy, C1- ***17*** ***alkyl***) into 10-65% of the amino groups on the surface of the enzyme with chems. such as RC(:O)OC6H4S(Me)2+CH3SO4-. In org. solvents, the modified enzymes are more stable than the wild type enzyme: therefore, they are useful for peptide synthesis. Modification of chymotrypsin with 4-benzyloxycarbonyloxyphenyl dimethylsulfonium methylsufate (Z-DSP) was shown. In synthesis of Z-Tyr-Gly-NH2 from benzyloxycarbonyl tyrosine and glycinamide in the presence of 50% DMF (dimethylformamide), the chymotrypsin having 35% of its amino group modified gave 64% prodn.; the unmodified enzyme, contrary, gave 5% prodn. Also given was the modification of trypsin, and again org. solvent-resistance of the modified enzyme was obsd.

L22 ANSWER 4 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1991:430756 CAPLUS
DN 115:30756
TI Abrasive tapes for finishing of magnetic recording heads
IN Sato, Masami; Fujiyama, Masaaki; Nishikawa, Yasuro; Iwasaki, Takashi
PA Fuji Photo Film Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 02095578	A2	19900406	JP 1988-247220	19880930
JP 2588006	B2	19970305		

AB The title tapes are prepd. by forming layers contg. abrasives, binders, and the polyethers R1Z(CH2CHR2O)nH (R1 = C6-30 hydrocarbyl, C1- ***17*** ***fluoroalkyl*** ; R2 = H, ***alkyl*** ; Z = O, NH, S, SO2, sulfonylimino; n = 1-30) on flexible substrates. A mixt. of powd. .alpha.-Fe2O3 225, Cr2O3 75, vinyl chloride polymer (contg. 3.5% epoxy group and 0.5% SO3Na group) 8.3, sulfonated polyurethane 4.8, 3:1 2,4-TDI-trimethylolpropane adduct 9.6, C16H33O(CH2CH2O)10H, MEK 100, and cyclohexane 100 parts was coated on a PET film and dried to give a film with a 5-.mu.m abrasive layer which could finish a magnetic head in 21 s; vs. 98 with Bu stearate instead of the polyether.

L22 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1988:196001 CAPLUS
DN 108:196001
TI Heat-sensitive recording material contg. phthalide derivative, fluorene derivatives and phenylenediamine derivatives
IN Kanda, Nobuo; Hirahara, Kazuko; Kondo, Mitsuru
PA Kanzaki Paper Mfg. Co., Ltd., Japan
SO Eur. Pat. Appl., 48 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 232907	A2	19870819	EP 1987-101874	19870211
EP 232907	A3	19880427		
EP 232907	B1	19910327		
R: DE, GB				
JP 62184881	A2	19870813	JP 1986-28098	19860212
JP 05016357	B4	19930304		
JP 63015785	A2	19880122	JP 1986-147775	19860624
JP 06045264	B4	19940615		
US 4761396	A	19880802	US 1987-11101	19870205

PRAI JP 1986-28098 19860212
JP 1986-147775 19860624
GI For diagram(s), see printed CA Issue.
AB A heat-sensitive recording material comprising .gtoreq.1 phthalide deriv I [R1-10 = H, halo, nitro, (substituted) alkyl, (substituted) cycloalkyl, (substituted) alkoxy, (substituted) acyloxy, (substituted) aryl, (substituted) phenoxy, (substituted) thioalkoxy, NR12R13 wherein R12, R13 = H, (substituted) alkyl, (substituted) cycloalkyl, (substituted) aryl, (substituted) aralkyl, tetrahydroflurfuryl, (substituted) acyl; R12 and R13 may form a hetero ring together or with an adjacent benzene ring; R11 = H, lower alkyl, a, b, c, and d represent C atoms and one or two of them may be N atom; the C atom may have a substituent selected from H, halo, alkyl, alkoxy, (substituted) amino, nitro; a-b, b-c, or c-d bond may form another arom. ring as a basic dye is claimed wherein .gtoreq.1 compd. selected from ***fluorene*** deriv. II [R14- ***17*** = C1-8 ***alkyl*** , C5-8 cycloalkyl, C3-8 alkoxyalkyl, aryl or aralkyl (un)esubstituted with halo, C1-4 alkoxy; R14R15 or R16R17 may form a hetero ring together or with an adjacent benzene ring; X = H, halo, C1-4 satd. alkyl, C1-4 alkoxyalkyl; NR18R19 wherein R18 and R19 are each same as R14-17; n = 1-4] and a p-phenylenediamine deriv. III [R20, R21 = C1-10 satd. alkyl, C3-9 unsatd. alkyl, C5-8 cycloalkyl, 3-methacryloyloxy-2-hydroxypropyl, aralkyl, aryl, arylsulfonyl (un)substituted with halo, C1-4 alkoxy] are further contained in the recording material. The recording material can be read by an optical character-reading device having a reading wavelength range over the IR region and has good preservability of the record images, particularly stable against humidity and heat.

L22 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1986:628976 CAPLUS
DN 105:228976
TI All-purpose sanitary cleaning composition
IN Wenzel, George
PA Sanitary Products Corp., USA
SO U.S., 4 pp. Cont. of U.S. Ser. No. 520,626, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 4612135	A	19860916	US 1985-765578	19850814
PRAI US 1983-520626		19830805		

AB A sanitary cleaning compn., capable of cleaning everything in restrooms, comprises H3PO4 0.12-20, oxalic acid 0.04-9, nonionic surfactant R(OCH2CH2)nOH (R = alkylaryl, n = 1-20) 0.1- ***17*** , cationic surfactant (***fluorinated*** ***alkyl*** quaternary ammonium iodide contg. C4-16 alkyl group) 0.03-0.3, glycol ether HOCH2CH2OR (R = lower alkyl) 0.14-44, and odor-masking agent 0.006-1.7, the balance being water and H3PO4 being present in excess of the oxalic acid. Thus, a cleaning compn. contained water 1830.0, 85% H3PO4 195.0, oxalic acid 67.5, BuOCH2CH2OH 200.0, ethoxylated nonylphenol 120.0, cationic surfactant (fluorinated alkyl quaternary ammonium iodide) 4.5, and odor-masking agent (mixt. of wintergreen, pine oil, geranium crystals, coumaric anhydride, and turpentine) 9.0 lbs.

L22 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1981:568956 CAPLUS
DN 95:168956
TI Some new membrane labeling agents having a benzacridine structure
AU Jacquignon, Pierre; Viallet, Pierre; Ricci, Adolfo; Bistocchi, Giovanni Alunni; De Meo, Giovanni; Vigo, Jean; Pedini, Mauro; Binaglia, Luciano
CS Inst. Chim. Subst. Nat., CNRS, Gif-sur-Yvette, 91190, Fr.
SO C. R. Seances Acad. Sci., Ser. 2 (1981), 292(8), 675-8
CODEN: CRSUDO
DT Journal
LA French
GI

/ Structure 2 in file .gra /

AB Dinaphthopyridines I (R = C1- ***17*** ***alkyl***), useful as ***fluorescent*** labeling agents for cell membranes (no data), were prepd. from N-(1-naphthyl)-2-naphthylamine (II). A mixt. of EtCO2H, II, and ZnCl2 was heated 3 h at 220.degree. to give I (R = Et).

L22 ANSWER 8 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1978:444639 CAPLUS
DN 89:44639
TI Heat stabilizers for halogen-containing resins
IN Tadenuma, Masahiko; Sato, Tamotsu; Tanaka, Kichiji; Shibatsuji, Takeo
PA Akishima Kagaku Kogyo K. K., Japan
SO Japan. Kokai, 8 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 53009847	A2	19780128	JP 1976-84378	19760715
JP 57039260	B4	19820820		

GI

/ Structure 3 in file .gra /

AB Halogen-contg. resins are stabilized with I (R = C1-17 alkyl, alkenyl, aryl, substituted aryl, aralkyl, cycloalkyl, heterocyclic residue; R1 = C1- ***17*** ***alkyl*** , alkenyl, ***fluoroalkyl*** , aryl, substituted aryl, aralkyl, cycloalkyl, PhO, heterocyclic residue; n = 1-5; M = Group IA, IIA, IIB metal, Zr, Sb). For example, a compn. from PVC [9002-86-2] 100, DOP 48, epoxidized soybean oil 2, Zn stearate 0.5, Ba stearate 1.0, and I (R = Bu, R1 = C17H35, n = 2, M = Zn) [66625-88-5] 0.1 part gave a transparent colorless sheet becoming light yellow after 40 min in a 180.degree. air oven, while a sheet not contg. I became yellowish red in 20 min.

L22 ANSWER 9 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1968:487366 CAPLUS
DN 69:87366
TI Hydrolysis of 9.alpha.-fluoro-11.beta.,21-dihydroxy-16.beta.-methyl-16.alpha.,17.alpha.-oxidopregna-1,4-diene-3,20-dione 21-acetate
IN Taub, David; Hoffsommer, Robert D., Jr.; Wendler, Norman L.
PA Merck and Co., Inc.
SO U.S., 3 pp. Continuation-in-part of U.S. 3285940
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 3378574	A	19680416	US 1966-538579	19660330

GI For diagram(s), see printed CA Issue.
AB The title compd. was prepd. from 9.alpha.-fluoro-11.beta.,21-trihydroxy-16.alpha.-methylpregna-1,4-diene-3,20-dione (I). I disemicarbazone, m. >300.degree., on acetylation with Ac2O in HOAc under reflux 1 hr. gave the 21-acetate. The reaction mixt. was treated with H2O and 5 hrs. on a steam bath to afford 9.alpha.-fluoro-11.beta.,21-dihydroxy-16-methylpregna-1,4,16-triene-3,20-dione 21-acetate (II), m. 228-33.degree.. Addn. of 10 g. Na2HPO4 to a stirred soln. of 510 mg. II in 15 ml. CH2Cl2, cooling to 0.degree., addn. of 2.5 moles 2M soln. peroxytrifluoroacetic acid, and reaction 1 hr. at room temp. gave 9.alpha.-fluoro-11.beta.,21-dihydroxy-16.beta.-methyl-16.alpha.,17.alpha.-oxidopregna-1,4-diene-3,20-dione 21-acetate (III). Addn. of 10 ml. 5% HCl in HOAc to 200 mg. III in 10 ml. HOAc and reaction for 5 min. afforded 9.alpha.-fluoro-11.beta.,17.alpha.,21-trihydroxy-16-methylpregna-1,4,15-triene-3,20-dione 21-acetate, 9.alpha.-fluoro-11.beta.,17.alpha.-21-trihydroxy-16-methylenepregna-1,4-diene-3,20-dione 21-acetate, 9.alpha.-fluoro-11.beta.,21-dihydroxy-16-methylpregna-1,4,14,16-tetraene-3,20-dione 21-acetate, and 9.alpha.-fluoro-15-chloro-11.beta.,21-dihydroxy-16-methylpregna-1,4,16-triene-3,20-dione 21-acetate. By using HBr in HOAc or HF in tetrahydrofuran, the corresponding 15-bromo and 9.alpha.,15-difluoro compds. were obtained.

L22 ANSWER 10 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1965:489178 CAPLUS
DN 63:89178
OREF 63:16429g-h,16430a-h,16431a-h,16432a-h,16433a-h,16434a-b
TI 10.alpha.-Methyl-9.beta.-hormonal steroids
IN Reerink, Engbert H.; Westerhof, Pieter; Schoeler, Hendrik F. L.
PA North American Phillips Co., Inc.
SO 56 pp.
DT Patent
LA Unavailable
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 3198792		19650803	US	19590408
DE 1228610			DE	

AB 4,7,22-Lumistatrien-3-one (125 g.) in 2.2 1. iso-PrOH previously satd. with dry HCl, dry HCl passed through the soln. for 0.5 hr., and worked up gave 80.5 g. lumista-4,6,22-trien-3-one (I), m. 101-2.degree. (ligroine), [.alpha.]25D -632.degree. (all rotations in CHCl3 unless otherwise mentioned). I (3 g.) in 300 ml. Et2O added to 450 ml. liquid NH3, treated with 420 mg. Li in NH3 and the product chromatographed on Al2O3 gave 2.43 g. lumista-4,22-dien-3-one (II), m. 122-4.degree. (Me2CO), [.alpha.]20D -125.degree.. II(20g.)in 750 ml. CH2Cl2 and 5.75 ml. C5H5N ozonized 4.5 hrs. at -80 gave 10.1 g. 3-oxo retrobisnor-4-cholen-22-al (III), m. 122-30.degree. (ligroine), [.alpha.]23D -144.degree.. III (450 mg.) in 15 ml. CHCl3 and 25 ml. AcOH oxidized 16 hrs. at 30.degree. with 200 mg. CrO3 and 0.2 ml. H2O gave 340 mg. 3-oxoretrobisnor-4-cholenic acid (IV), m. 202-4.degree. (Et2O). Alternatively a soln. of 450 mg. of an ozonide of II in 25 ml. CH2Cl2 left overnight at 30.degree. with 200 mg. CrO3 in 25 ml. AcOH gave 150 mg. IV. III (300 mg.), 0.11 ml. piperidine, and 1 to 5 mg. p-MeCoH4SO3H refluxed 3 hrs. in 5 ml. dry C6H6 gave 185 mg. 22-(N-piperidyl)retrobisnorchola-4,20(22)dien-3-one (V), m. 94-6.degree. (MeOH). Other samples of V m. 114-15.degree. (probably due to cis-trans isomerism). Alternatively 10 g. III refluxed 3 hrs. in 180 ml. C6H6 with 3.8 ml. piperidine and 30 mg. p-MeC6H4SO3H gave 9.3 g. V. III (1 g.) refluxed with 0.5 g. fused NaOAc and 50 ml. Ac2O gave 22-acetoxyretrobisnorchola-4,20(22)-dien-3-one (VI). V (300 mg.) in 4.5 ml. C6H6 added in 45 min. at -5.degree. to +5.degree. to 453 mg. Na2Cr2O7.2H2O in 4.5 ml. AcOH and 3 ml. C6H6 and the product worked up gave 150 mg. retroprogesterone (VII), m. 163-4.degree. (CH2Cl2-ligroine), [.alpha.]20D

- 62.degree.. III treated with NaOAc in Ac2O gave VI and VI ozonized, decompd., and hydrolyzed gave VII. VII (10 g.) in 79 ml. C6H6 stirred 1.5 hrs. with NaOMe in diethyl oxalate and some C6H6 gave 10.8 g. Na enolate of 21-ethoxyoxalylretroprogesterone (VIII). VIII in 150 ml. MeOH treated 40 min. at -20 with 5.9 g. iodine in 210 ml. MeOH, mixt. stirred 1.5 hrs., and the iodine compd. decompd. with NaOMe soln. gave 5.55 g. 21-iodoretroprogesterone (IX). IX (5.55 g.) refluxed 18 hrs. in 200 ml. Me2CO with 12 g. KOAc and chromatographed on Al2O3 gave 335 mg. retrodeoxycorticosterone acetate, m. 165-8.degree. (alc.). V (9.6 g.) in 475 ml. CH2Cl2 treated dropwise at -55.degree.with 4.08 g. Br in 50 ml. CH2Cl2, the dibromo compd. stirred 2 hrs. at 20.degree., the 20-bromo deriv. heated 1 hr. at 70.degree. with 70 ml. C5H5N, then 0.5 hr. at 100.degree., and worked up gave 6 g. 3-oxoretrobisnorchol-4,17(20)-dien-22-al (X), m. 155-9.degree. (Me2CO-alc.), [.alpha.]23D -138.degree.. III (7.7 g.) in 100 ml. CCl4 treated with 48 ml. Br soln. in CCl4 (0.515 mole/ml.) and 3 g. CaCO3 and then treated with C5H5N gave 7.15 g. X. X (5 g.) suspended with 8 g. NaCN in 50 ml. MeOH at -20.degree., left 2 hrs. at 20.degree. with 7.1 ml. AcOH, then 40 hrs. at 5.degree., and worked up gave the 22-HCN addn. product of X which treated at -80.degree. with O3 gave 1.48 g. retroandrost-4-en-3,17-dione (XI), m. 154-6.degree. (alc.). XI (3.03 g.) in 25 ml. C6H6 and 25 ml. Et2O left 16 hrs. with 1.61 g. K in liquid NH3 which had been treated with CH.tplbond.CH gave 1.95 g. 17.alpha.-ethynylretrotestosterone (XIa), m. 195-6.degree. (hexane), [.alpha.]20D -219. 3-Oxoretrobisnorchola-4,6-dien-22-al (XII) (3.5 g.) in 50 ml. C6H6 refluxed 2.5 hrs. with 1.27 ml. piperidine and 20 mg. p-MeC6H4SO3H gave 2.1 g. 22-(N-piperidyl)retrobisnorchola-4,6,20(22)-trien-3-one (XIII), m. 135-6.degree. (Me2CO). XIII oxidized with Na2Cr2O7 in AcOH gave 6-dehydroretroprogesterone (XIV), m. 168-9.degree. (Me2CO). Lumisterone (3.95 g.) in 150 ml. CH2Cl2 and 0.81 ml. C5H5N treated with O3 gave 3.11 g. 3-oxoretrobisnorchola-4,7-dien-22-al (XV), m. 196-200.degree. (CH2Cl2-Me2CO). XV treated with piperidine and then oxidized with Na2Cr2O7 gave 7-dehydroretroprogesterone (XVI). By isomerization of the 3-oxo-4,7-dehydro system of XVI with dry HCl XIV was obtained. VII (7.5 g.) in 500 ml. tert-BuOH refluxed 5 hrs. with 12.75 g. chloranil and the product chromatographed on Al2O3 gave XIV. I (3.95 g.) ozonized as described above gave 3.08 g. XII, m. 153-5.degree. (Me2CO). 3-Oxoretrobisnorchol-4,20(22) dien-22-al (0.978 g.) in 10 ml. C6H6 kept 4 hrs. at 0.degree. with 1.2 g. monoperphthalic acid in 25.5 ml. EtOAc gave 0.88 g. 17(20)epoxy-20-formyloxyretropregn-4-en-3-one (XVII). Hydrolysis of XVII with 2N NaOH gave 17.alpha.-hydroxyretroprogesterone (XVIII), m. 222-5.degree. (alc.). XIII (6.5 g.) treated with Br and then with C5H5N gave 2.41 g. retrobisnorchola-4,6,17(20)-trien-3-one-22-al (XIX), m. 217-19.degree. (Me2CO). XIX (15 g.) in 150 ml. EtOAc and 150 ml. C6H6 treated with 20.2 g. monoperphthalic acid in 450 ml. EtOAc and left 16 hrs. gave 16.4 g. resinous epoxy-20-formyloxy compd. which treated 1.5 hrs. at 30.degree. with 2N NaOH gave 5.69 g. 6-dehydro-17.alpha.-hydroxyretroprogesterone (XX). XIX (3.7 g.) treated as above with 5.9 g. NaCN and MeOH followed by ozonization gave 1.2 g. retroandrosta-4,6-diene-3,17-dione, m. 189-90.degree. (Me2CO). XVIII (220 mg.) and 220 mg. p-MeC6H4SO3H in 15 ml. AcOH kept 18 hrs. at room temp. gave 130 mg. 3,17-diacetate of XVIII, m. 217-18.degree. (CH2Cl2-MeOH). XVIII (0.5 g.) similarly treated but in less Ac2O and the product fractionally crystd. gave 260 mg. 17.alpha.-acetoxyretroprogesterone, m. 171-3.degree. (MeOH). XVIII (900 mg.) treated with caproic anhydride and p-MeC6H4SO3H, then with 0.3 ml. concd. HCl in 20 ml. alc., and the product chromatographed gave 67 mg. 17-caproate of XVIII, m. 50-3.degree. (hexane). Many esters of XVIII were similarly prepg. XVIII (0.495 g.) in tert-BuOH treated with chloranil gave 50 mg. XX, m. 242-5.degree. (alc.-hexane). 6-Dehydro-17.alpha.-acetoxyretroprogesterone, m. 181-3.degree., was similarly prepd. Retroprogesterone hydroxylated with Rhisopus nigricans gave a hydroxyretroprogesterone, m. 217-18.degree., and 17.alpha.,21-dihydroxyretroprogesterone (XXa) incubated with Aspergillus ochraceus gave retrohydrocortisone, m. 269.degree. (decompn.). XIa (1.88 g.) in 50 ml. C5H5N hydrogenated over 2 g. Pd-CaCO3 gave 1.17 g. 17.alpha.-vinylretrotestosterone, m. 143.5-5.5.degree. (Me2CO-hexane). VII (5 g.) in 250 ml. C6H6 refluxed 7 hrs. with 4 g. dichlorodicyanobenzoquinone and the product chromatographed on silica gel gave 2.14 g. 1-dehydroretroprogesterone, m. 154-5.5.degree. (Me2CO-hexane). XIV (2.5 g.) gave 208 mg. dioxime, m. 279-82.degree. (tetrahydrofuran-ligroine). I (75 g.) and 55 ml. HCO2Et in 1.2 l. C6H6 kept 3 days at room temp. with 16.5 g. NaH and the Na salt converted to the free alc. gave 2-hydroxymethylenelumista-4,6,22-trien-3-one (XXI) as the hydrate, m. 119-22.degree., anhydrous form m. 122-3.5.degree.. Similarly, II gave 2-hydroxymethylenelumista-4,22-dien-3-one (XXII), m. 134-5.degree.. XXI (108 g. of Na salt) in 3.2 l. alc. treated 3.5 to 4.5 hrs. at 10.degree. with perchloryl fluoride gave 71 g. 2-fluorolumista-4,6,22-trien-3-one (XXIII), m. 158-60.degree. (MeOH), [.alpha.]27D -635.degree. (alc.). XXII similarly gave 2-fluorolumista-4,22-dien-3-one. XXIII (30.5 g.) in 610 ml. CH2Cl2 and 10 ml. C5H5N treated with O3 at -80.degree. gave 16.6 g. 2-fluororetrobisnorchola-4,6-dien-3-on-22-al (XXIV), m. 183-5.degree. (CH2Cl2-Et2O). XXIV (2.08 g.) similarly treated with piperidine and p-MeC6H4SO3H gave 2-fluoro-22-(N-piperidyl)-retrobisnorchola-4,6,20(22)-trien-3-one, which with CrO3 gave 2-fluoro-6-dehydroretroprogesterone, m. 153-4.degree. (alc.). Retropregnane-3,20-dione (XXV) (300 mg.) in alc. treated 3 hrs. at -20.degree. with 36.6 mg. CaCl2.2H2O and 16.3 mg. NaBH4 gave retropregnan-3-ol-20-one, m. 167-71.degree. (alc.-E+2O). VII (2.826 g.) in 150 ml. dioxane shaken with excess H over 0.3 g. 10% Pd-C in the presence of 0.6 g. KOH in 9 ml. MeOH gave XXV, m. 115-16.degree. (E+2O-hexane). XIV (5 g.), 7.5 g. chloranil, and 25 g. CaCO3 refluxed 3 hrs. with 170 ml. isoamyl alc. and chromatography of the product gave XIV and 150 mg. 1,6-bisdehydroretroprogesterone (XXVI). XIV (5 g.) and 5.1 g. dihydroquinone in 250 ml. C6H6 refluxed 6 hrs. gave 1.52 g. XXVI, m. 143-3.5.degree. (Me2CO-hexane). VII enol acetate (7.5 g.) and dioxane treated at room temp. with perchloryl fluoride gave 185 mg. 6.alpha.-fluororetroprogesterone (XXVII), m. 150-1.degree. (Me2CO-hexane), 1.16 g. 6.beta.-fluororetroprogesterone (XXVIII), m. 163-5.degree. (MeOH), and 77 mg. 6-hydroxyretroprogesterone, m. 220-2.degree. (Me2CO). XXVII (50 mg.) in 10 ml. CHCl3 treated 1 hr. with passage of dry HCl gave XXVIII. XVIII (1 g.) in 15 ml. tetrahydrofuran and 2.5 ml. MeOH treated at -4.degree. to 0.degree. with 1.5 g. iodine and the product refluxed with KOAc in Me2CO gave 772 mg. 17.alpha.,21-dihydroxyretroprogesterone 21-acetate (XXIX), m. 218-238.degree. (decompn.) (Me2CO). XX (1 g.) in 15 ml. tetrahydrofuran and 2.5 ml. MeOH similarly treated with iodine and the product refluxed with KOAc in Me2CO gave 915 mg. 6-dehydro-17.alpha.,21-dihydroxyretroprogesterone 21-acetate, m. 238.5-44.degree. and 257-9.degree. (decompn.). XXIX (1.2 g.) in 30 ml. MeOH stirred 2 hrs. at 24.degree. with 216 mg. K2CO3 in 6 ml. H2O gave 897.5 mg. XXa. XIa (3.12 g.) in 250 ml. dioxane reduced with H over PdCaCO3 and the product chromatographed on silica gel gave 300 mg. 17.alpha.-ethyl-5-retroandrostan-17-ol-3-one (A isomer), m. 125-6.5.degree. (Et2O-petr. ether) and 828 mg. B isomer, m. 125-6.5.degree.. VII was converted to its 3-enol acetate, m. 90-2.degree. (MeOH). Retroandrosta-4,6-diene-3,17-dione (2.35 g.) in 70 ml. 2:1 mixt. C6H6-Et2O stirred 0.5 hr. at -80.degree. with 1.28 g. K in 50 ml. liquid NH3 previously treated with CH.tplbond.CH gave 6-dehydro-17.alpha.-ethynylretrotestosterone, m. 205-7.degree. (Me2CO-hexane). IX (6.6 g.) in 200 ml. MeCN and 5.72 g. AgF stirred 25 hrs. at 45.degree. gave 21-fluororetroprogesterone, m. 173-5.degree. (alc.-Me2CO). XIV (10 g.) in 70 ml. C6H6 stirred 1.5 hrs. with 8.5 ml. (CO2Et)2 and 10.5 ml. of a 3.45N NaOMe soln. in MeOH, 70 ml. C6H6, and 3.2 ml. alc. gave 12.2 g. Na enolate of 21-ethoxyoxalyl-6-dehydroretroprogesterone (XXX). XXX in 170 ml. MeOH treated at -25.degree. with 7.2 g. iodine gave 12-iodo-6-dehydroretroprogesterone (XXXI). XXXI refluxed 5 hrs. with KOAc in Me2CO, H2O, and AcOH gave 6-dehydro-21-acetoxyretroprogesterone, m. 194.5-7.5.degree.. XIV (937 mg.) in 125 ml. CCl4 treated in 50 min. with 0.5 g. Br in 10 ml. CCl4 gave 4-bromo-6-dehydroretroprogesterone m. 121-2.degree. (MeOH) (decompn.). XIV with Cl similarly afforded 4-chloro-6-dehydroretroprogesterone, m. 185-6.degree.. 6-Chlororetroprogesterone (XXXII) (700 mg.) and 1 g. chloranil refluxed 30 hrs. in 100 ml. tert-BuOH gave 6-chloro-6-dehydroretroprogesterone, m. 165-6.degree. (alc.). 3-Acetoxyretropregna-3,5-dien-20-one (XXXIII) (1.85 g.) in 30 ml. Et2O, 4 g. KOAc, 60 ml. 85% AcOH treated with 375 mg. Cl in 9.4 ml. AcOH gave XXXII, m. 197-9.5.degree. (EtOAc), and 3,6,20-trioxoretropregnane, m. 201-3.degree.. 6-Bromoretroprogesterone was similarly prepd., m. 138-40.degree. (decompn.). XIV (5 g.) reduced with NaBH4 in alkali gave retropregna-4,6-diene-3,20-diol (XXXIV). XXXIV in C6H6 refluxed with 50 g. MnO2 gave 3.27 g. retropregna-4,6-dien-20-ol-3-one, m. 198-8.5.degree. (Me2CO), and a second C-20 isomer, m. 173-5.degree.. Retropregna-4-en-20-ol-3-one (XXXV) treated with NaH and HCO2Et and the Na product acidified with 5% HCl gave 2-hydroxymethyleneretropregn-4-en-20-ol-3-one, m. 94-9.degree. (Et2O), [.alpha.]21D -155.degree. (alc.). VII (2 g.) in 40 ml. tetrahydrofuran reduced with LiAlH4 gave retropregn-4-ene-3,20-diol and this product refluxed with MnO2 in C6H6 gave 1.69 g. XXXV, m. 174-6.degree. (Me2CO-hexane). XIV (1 g.) in 10 ml. CH2Cl2 treated with alc.-HCl gave 3-ethoxyretropregna-3,5,7-trien-20-one, m. 115-16.degree. (MeOH). XXXII (7.5 g.) in 200 ml. AcOH refluxed 4.5 hrs. with 21 g. KOAc gave 2-acetoxyretroprogesterone, m. 195.5-7.0.degree.(alc.). XXXIII treated with monoperphthalic acid in EtOAc gave 6-hydroxyretroprogesterone (XXXVI). Acetylation of XXXVI gave 6-acetoxyretroprogesterone, m. 177-8.5.degree. (alc.). XX was converted into the 17-caproate, m. 110-12.5.degree. (E2O-hexane). 3,17-Diacetoxyretropregna-3,5-dien-20-one (13.2 g.) in 950 ml. dioxane and 40 ml. H2O treated 1 hr. at room temp. with passage of perchloryl fluoride gave 6.alpha.-fluoro-17.alpha.-acetoxyretroprogesterone, m. 177-9.degree. (Me2CO-hexane), and 6.beta.-fluoro-17.alpha.-acetoxyretroprogesterone, m. 197.5-8.5.degree. (Me2CO), plus 3,6,20-trioxoretropregnan-17.alpha.-ol 17-acetate, m. 241-4.degree. (Et2O-C6H6-Me2CO). XIV (25 g.) treated with BzO2H gave 6,7-oxidoretroprogesterone (XXXVII), m. 187-90.degree. (EtOAc), and some 6-chloro-7-hydroxyretroprogesterone (XXXVIII) due to splitting of the epoxy bond of XXXVII during chromatography. XXXVIII treated with HCl 3 hrs. at room temp. gave 6-chloro-6-dehydroretroprogesterone. XXVIII (4 g.) and 4 g. dichlorodicyanobenzoquinone in C6H6 refluxed 7.5 hrs. over 0.4 g. p-nitrophenol gave 2 g. 1-dehydro-6.beta.-fluororetroprogesterone, m. 208-11.degree. (EtOAc). 17.alpha.-Acetoxyretroprogesterone in MeOH treated 6 days with CuBr gave 6-dehydro-6-methoxy-17.alpha.-acetoxyretroprogesterone, m. 275-80.degree. (alc.). XIV (3 g.) in 3 ml. thioacetic acid refluxed 2.5 hrs. gave 7-acetylthioretroprogesterone, m. 133-5.degree. (Me2CO-hexane). VII similarly treated with CuBr gave 6-dehydro-6-methoxyretroprogesterone, m. 206-8.degree.. XI (3 g.) in 30 ml. MeOH refluxed 10 min. with 1.6 ml. pyrrolidine gave 3.31 g. 3-(N-pyrrolidino)retroandrosta-3,5-dien-17-one (XXXIX). XXXIX (3.3 g.) refluxed 5 hrs. with a soln. contg. methallyl-magnesium chloride and worked up gave 2.52 g. 17.alpha.-(2-methallyl)retrotestosterone, m. 91-3.degree. (MeOH). Dried sample from Et2O m. 106-8.degree.. I (60 g.) and 6 g. CuCl in 500 ml. tetrahydrofuran stirred 3 hrs. with soln. prepd. from 100 ml. MeBr, 25 mg. Mg, and 5 g. CuCl in 200 ml. tetrahydrofuran gave 29 g. 7-methyllumista-4,22-dien-3-one (XL), m. 94.5-5.5.degree. (ligroine), [.alpha.]22D -150.5.degree.. XL upon ozonization gave 3-oxo-7-methylretrobisnorchol-4-en-22-al (XLI), m. 161.5-2.0.degree., [.alpha.]23D -148. XLI (4.65 g.) in 100 ml. C6H6 refluxed 5 hrs. with 2 ml. piperidine and 2 mg. p-MeC6H4SO3H gave 7-methyl-22-(N-piperidyl)-retrobisnorchola-4,20(22)-dien-3-one (XLII), m. 153.5-4.5.degree., [.alpha.]23D -120.degree.. XLII (1.96 g.) treated with Na2CR2O7 in AcOH gave 7-methylretroprogesterone, m. 206-8.degree., [.alpha.]25D -57.degree.. XXXIX treated with CH2:CHCH2MgCl gave 17.alpha.-

allylretrotestosterone, m. 74-8.degree.. XX (1 g.) heated 45 min. at 80.degree. with 3.3 g. trimethylacetic acid and 1 ml. trifluoroacetic anhydride gave 6-dehydro-17.alpha.-hydroxyretroprogesterone 17-pimalate, m. 214-16.degree.. 17.alpha.-Acetoxyretroprogesterone (4 g.) refluxed 15 hrs. with 2.7 g. dichlorodicyanobenzoquinone in 200 ml. C6H6 gave 1 -dehydro-17.alpha.-acetoxyretroprogesterone, m. 183-4.5.degree.. XIa treated with Ac2O-p-MeC6H4SO3H and then with concd. HCl in MeOH gave 17.alpha.-ethynylretrotestosterone 17-acetate, m. 183-4.degree. (Me2CO). VII by hydroxylation (microbiol.) gave 16.alpha.-hydroxyretroprogesterone (XLIII), m. 172.5-4.5.degree., [.alpha.]25D -92.3.degree.. Dehydration of XLIII gave 16-dehydro retroprogesterone, m. 165-7.degree.. Microbiol. hydroxylation of VII gave 15.alpha.-hydroxyretroprogesterone, m. 203-5.degree., [.alpha.]D -23.degree.. Oxidn. of 11,17.alpha.,21-trihydroxyretroprogesterone 21-acetate with CrO3 gave retrocortisone 21-acetate, m. 275.degree. (decompn.). Microbiol. hydroxylation of XIV gave 6-dehydro-16.alpha.-hydroxyretroprogesterone (XLIV), m. 200-3.degree., [.alpha.]D -526.degree.. Dehydration of XLIV gave 6,16-bisdehydroretroprogesterone, m. 163-5.degree.. Hydroxylation of VII gave 11-hydroxyretroprogesterone (XLV). Oxidn. of XLV with CrO3 gave 11-oxoretroprogesterone, m. 158-60.degree.. Hydroxylation of XVIII gave 11,17.alpha.-dihydroxyretroprogesterone (XLVI), m. 202-6.5.degree. (decompn.), [.alpha.]D -118.degree.. XLVI oxidized as above gave 11-oxo-17.alpha.-hydroxyretroprogesterone, m. 240-5.degree.. XXIII was converted into 2-fluoro-6-dehydro-17.alpha.-acetoxyretroprogesterone, m. 204-5.degree.. Degradation of the side-chain of 2-fluorodihydroisolumisterone gave 2-fluororetroprogesterone, m. 162-4.degree.. II was converted to 2-(ethoxyoxalyl)lumista-4,22-dien-3-one then to 2-methyllumista-4,22-dien-3-one (XLVII). XLVII ozonized, treated with Ac2O, and oxidized gave 2-methylretroprogesterone, m. 126-7.degree.. Degradation of the side chain of 2-methyllumista-4,6,22-trien-3-one gave 2-methyl-6-dehydroretroprogesterone, m. 168.5-70.0.degree.. Retroandrost-4-en-17-one (XLVIII) treated with CH2:CHCH2MgCl gave 17.alpha.-allylretroandrost-4-en-17-ol, m. 79-86.degree.. XLVIII with KC.tplbond.CH in iso-PrOH gave 17.alpha.-ethynylretroandrost-4-en-17-ol, m. 74-5.degree.. Treatment of the Na enolate of the 21-ethoxy oxalate of XXVI with perchloryl fluoride in MeOH and NaOMe gave after refluxing with KOAc 21-fluoro-1,6-bisdehydroretroprogesterone, m. 154-5.degree.. XI (1.13 g.) reduced with 550 mg. LiAlH4 gave retroandrost-4-en-3,17.beta.-diol (XLIX), m. 117-18.degree. (ligroine). Crude XLIX shaken 17 hrs. in 60 ml. CHCl3 with 6 g. MnO2 gave retrotestosterone (L), m. 115-6.degree. (E+2O), [.alpha.]23D -154.degree.. L gave the .beta.-phenylpropionate, m. 73-4.degree. (MeOH). L (5 g.) treated as above with chloranil 1.43 g. 6-dehydroretrotestosterone (LI), m. 174-5.degree. (E2O). LI (360 mg.) afforded 233 mg. propionate, m. 115-17.degree. (MeOH). L (0.5 g.) treated with 0.5 g. Li in 75 ml. NH3 and 50 ml. E2O and the product esterified gave 270 mg. bis(3,5-dinitrobenzoate) of retro-5-androstane-3,17.beta.-diol, m. 237-42.degree.(CH2Cl2-Me2CO). L (1 g.) in 35 ml. C6H6 refluxed 48 hrs. with 1 g. SeO2 and 0.6 ml. H2O and the product chromatographed on Al2O3 gave 98 mg. 1-dehydroretrotestosterone, m. 175-7.degree. (hexane-CH2Cl2). L (1.14 g.) was converted into enol of 2-(ethoxyoxalyl)retrotestosterone (LII). LII (1.3 g.) refluxed 18 hrs. with 2 ml. MeI in 25 ml. Me2CO and 1 g. K2CO3 and chromatographed on Al2O3 gave 207 mg. 2-methylretrotestosterone, m. 177-9.5.degree. (Et2O-hexane). Retroandrosta-4,6-dien3,17-dione (LIIa) treated with LiAlH4 gave retroandrosta-4,6-diene-3,17.beta.-diol (LIII), m. 140-4 (decompn.). Crude LIII refluxed 30 hrs. with MnO2 in C6H6 gave 3 g. LI. 6-Bromoretrotestosterone 17-acetate treated with C5H5N gave 17-acetate of LI, m. 131-3.degree. L (1 g.) and 0.005 ml. concd. H2SO4 refluxed 3 hrs. with 5 ml. isopropenyl acetate gave 0.9 g. 3,17.beta.-diacetoxyretroandrosta-3,5-diene, m. 118-19 (MeOH). L (1g.)added to 0.4 g. K in 20 ml. tert-BuOH, left 3.5 hrs. at room temp. with 1.3 ml. MeI gave 4,4-dimethylretroandrost-5-en-17.beta.-ol-3-one, m. 152.5-3.5.degree.. L gave the p-hexyloxyphenylpropionate and propionate (LIV), m. 107-8.degree.. LIV acetylated with isopropenyl acetate in the presence of traces of concd. H2SO4 gave 3-acetoxyretroandrosta-3,5-dien-17.beta.-ol 17-propionate, m. 102-4.degree.. 6-Bromoretrotestosterone 17-acetate (410 mg.) and 1.3 g. KOAc in 10 ml. AcOH refluxed 4 hrs. gave 2-hydroxyretrotestosterone 2,17-diacetate, m. 184-6.degree.. 3,17-Diacetoxyretroandrosta-3,5-diene treated with KOAc in AcOH, then with Br in AcOH, and worked up gave 6-bromoretrotestosterone 17-acetate, m. 130.degree. (decompn.). 3,17.degree.-Diacetoxyretroandrosta-3,5-diene (4 g.) in 178 ml. EtOAc contg. 14.3 mg. monoperphthalic acid/ml. left overnight at 5.degree. gave mixt. (LV) of 6.alpha.- and 6.beta.-hydroxyretrotestosterone 17-acetate, m. 167-70.degree.. LV (2.6 g.) in 10 ml. C5H5N and 10 ml. Ac2O left 20 hrs. at room temp. and the product separated gave one isomer of 6,17-diacetoxyretrotestosterone, m. 178-80.degree., and the other isomer, m. 116-17.degree.. LI was converted to the 17-palmitate, m. 58.5-9.0.degree.. L treated with NaH and HCO2Et and then with 5% HCl gave 2-hydroxymethyleneretrotestosterone (LVI), m. 98-120.degree. (aq. alc.). LVI (5 g.) in 13 ml. alc. refluxed 3 hrs. with 2.5 ml. N2H4.H2O in 40 ml. alc. gave 3.5 g. 17.beta.-hydroxyretroandrost-4-eno[3,2-c]pyrazole, m. 259-63.degree., [.alpha.]24D -149.degree. (alc.). LI (2 g. (in 10 ml. pyridine left 18 hrs. at room temp. with 2.5 g. p-hexyloxyphenylpropionyl chloride gave 3.9 g. resinous 6-dehydroretrotestosterone 17-(p-hexyloxyphenylpropionate). LI treated 16 hrs. at 60.degree. with succinic anhydride gave the 17-hemisuccinate, m. ***178*** -93.5-201. ***5*** -203.5.degree.. LI also gave the 17-phenylpropionate, m. 93-4.degree. (MeOH). LIIa treated with Br in CCl4 gave 4-bromoretroandrosta-4,6-dien-3,17-dione, m. 130-50.degree.. L 17-propionate (1.2 g.) in 150 ml. C6H6 treated with 9 ml. (CH2OH)2 gave 3,3-ethylenedioxyretroandrosta-3,5-dien-17.beta.-ol-17-propionate, m. 66-8.beta.. XXXIX (3 g.) treated with MeMgBr gave 17.alpha.-methylretrotestosterone (LVII), m. 133-4.degree.. LIIa similarly gave 3-ethoxyretroandrosta-3,5,7-trien-17-one, m. 118-19.5.degree.. LIIa (1 g.) in 15 ml. CH2Cl2 left 0.5 hr. at 0.degree. with dry HCl in MeOH gave 404 mg. 3-methoxyretroandrosta-3,5,7-trien-17-one, m. 139-40.degree.. L (2.5 g.) converted to the 17-hexahydrobenzoate, m. 71-3.degree. (petr. ether). LVII (2 g.) and dichlorodicyanobenzoquinone in C6H6 refluxed 7 hrs. gave 1-dehydro-17.alpha.-methylretrotestosterone, m. 163-4.degree.. XI treated with isopropenyl acetate gave 3-acetoxyretroandrosta-3,5-dien-17-one, m. 142-3.degree.. L (1.564 g.) in 6.25 ml. C5H5N treated 24 hrs. at room temp. with 1.56 g. p-toluenesulfonyl chloride gave the 17-tosylate, m. 164.5-5.5.degree.. The tosylate treated with KOAc in HCONMe2 and the resin hydrolyzed with KOH soln. gave retroandrosta-4,16-dien-3-one and L. L was converted into the 17-acetate, m. 128.5-30.0.degree.. 3,17.beta.-Diacetoxyretroandrosta-3,5-diene (10.3 g.) treated in dioxane with perchloryl fluoride and chromatographed on silica gel gave 6.alpha.-fluororetrotestosterone 17-acetate, m. 129.5-30.5.degree.. Microbiol. hydroxylation of L gave 16.alpha.-hydroxyretrotestosterone, m. 210-12.degree., [.alpha.]D -167.degree.. XXIII converted into 2-fluororetrobisnorchol-3-one-4-en-22-al, then into 2-fluororetrobisnorchol-3-one-4,17(20)-dien-22-al, 2-fluororetroandrost-4-en-3,17-dione, 2-fluororetroandrost-4-en-3,17-diol, 2-fluororetrotestosterone, and finally 2-fluoro-6-dehydroretrotestosterone, m. 90-5.degree. and 142-3.5.degree.. XI reduced with NaBH4 in alc.-H2O gave 3,17.beta.-dihydroxyretro-5-androst-4-ene and 8,17.beta.-dihydroxyretroandrostane (LVIII), m. 156-8.degree.. LVIII oxidized with CrO3 in Me2CO contg. H2SO4 gave retro-5-androstane-3,17-dione, m. 114-15.5.degree.. 17.alpha.-Methylretroandrost-4-en-17-ol was obtained by treatment of retroandrost-4-en-17-one (LIX) with MeMgI. L (2 g.) in 3 ml. Et2O and 6.5 ml. AcOH stirred 1 hr. with 0.72, ml. 1,2-ethanedithiol and 0.85 ml. BF3.Et2O gave 3-ethylene dithioketal of L, m. 166-7.5.degree.. This ketal (0.5 g.) in 2.5 ml. tetrahydrofuran added to 15 ml. liquid NH3 and 5 ml. tetrahydrofuran, stirred 15 min. with 0.4 g. L gave 3-deoxyretrotestosterone (LX). LX oxidized with CrO3 in Me2CO gave LIX, m. 89-90.5.degree.. L with AcCl gave the 17-acetate, m. 113-14.50. XLII (1 g.) in 50 ml. CH2Cl2 treated in 20 min. at -55.degree. with 0.15 ml. Br in 6 ml. CH2Cl2, mixt. heated to 0.degree., and product heated with C5H5N for 1 hr. at 70.degree. gave 7-methylretrobisnorchola-4,17(20)-dien-3-on-22-al (LXI), m. 182-3.5.degree.. LXI suspended in MeOH treated with NaCN, AcOH, and MeOH 2 hrs. from -20.degree. to +5.degree. and kept 40 hrs. at 5.degree. gave 7-methylretroandrost-4-ene-3,17-dione (LXII), m. 196-7.degree.. LXII was reduced to give 7-methylretroandrost-4-ene-3,17.beta.-diol (LXIII), m. 80-95.degree.. LXIII oxidized as above gave 7-methylretrotestosterone, m. 146.5-50.0.degree.. 3,17.beta.-Diacetoxyretroandrosta-3,5-diene (3 g.) in 60 ml. Et2O treated with 6.4 g. KOAc in 120 ml. 85% AcOH, and treated 5-10 min. at 0.degree. with 0.57 g. Cl gave 6-chlororetrotestosterone 17-acetate, m. 176-8.degree.. II treated with Et orthoformate in C6H6 and alc. gave 3-ethoxylumista-3,5,22-triene (LXIV), m. 76-7.5.degree. (alc.). LXIV (12.7 g.) in 60 ml. dioxane and 4.8 ml. C5H5N left 45 hrs. at room temp. with 20 g. CBr4 gave 7.46 g. 6-tribromomethyldihydroisolumisterone (LXV), m. 132-3.5.degree. (Me2CO-MeOH). LXV (100 mg.) in 15 ml. alc. heated with 15 ml. of a strong anion exchange resin gave 6-dibromomethylenedihydroisolumisterone, m. 100-2.5.degree., [.alpha.]23D 10.degree.. XI was converted into 3-(1-pyrrolidino)retroandrosta-3,5-dien-17-one (LXVI). LXVI treated with CH2:CMeCH2MgCl gave 17.alpha.-(2-methallyl)retrotestosterone, m. 106-8.degree.. Similarly LXVI gave 17.alpha.-allylretrostesterone, m. 76-8.degree.. Retroandrosta-4,6-diene-3,17-dione was converted into 3,7-di(1-pyrrolidino)retroandrosta-3,5-dien-17-one, and then alkylated to give 6-dehydro-17.alpha.-(2-methallyl)retrotestosterone. Many related compds. were prepd. by the above described procedures.

L22 ANSWER 11 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1964:3413 CAPLUS
DN 60:3413
OREF 60:602g-h,603a
TI 6- ***Fluoro*** - ***17*** .alpha.- ***alkyl*** -1,4-androstadien-17.beta.-ol-3-ones
IN Campbell, J. Allan; Pederson, Raymond L.; Babcock, John C.; Hogg, John A.
PA Upjohn Co.
SO 3 pp.
DT Patent
LA Unavailable

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 1139838		19621122	DE	
PRAI US		19571129		

GI For diagram(s), see printed CA Issue.
AB The title compds., superior gonadotropic agents, were prepd. from the corresponding 4-enes by dehydrogenation (fermentative, e.g. with Septomyxa affinis, or chem., e.g. with SeO2), followed by acylation and (or) epimerization to the 6.alpha.-fluoro compd., as exemplified by 6.alpha.-fluoro-17.alpha.-methyl-1,4-androstadien-17.beta.-ol-3-one (I), m. 161-63.degree. (CH2Cl2-hexane).

L22 ANSWER 12 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1962:469449 CAPLUS
DN 57:69449
OREF 57:13843e-i,13844a-c
TI 17.alpha.,21-Epoxypregnenes
IN Spero, George B.; Lincoln, Frank H., Jr.; Schneider, William P.
PA Upjohn Co.

SO 3 pp.
DT Patent
LA Unavailable

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 3012028		19611205	US	19590526

AB A cold soln. of 85 mg. MeSO2Cl in 1 ml. of pyridine was added to 200 mg. of 6.alpha.-methyl-9.alpha.-fluorocortisone in 1 ml. pyridine at 0.degree.. This mixt. was kept 3 hours between 0.degree. and 5.degree.. Ice and sufficient dil. HCl to neutralize the pyridine was added and the mixt. extd. with CH2Cl2. The exts. were washed with cold NaHCO3 soln, dried, and evapd. under reduced pressure to give 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.,21-dihydroxy-4-pregnene-3,11,20-trione 21-methanesulfonate (I). Similarly 1-dehydro-6.alpha.-methyl-9.alpha.-fluorocortisone gave 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.,21-dihydroxy- 1, 4-pregnadiene-3,11,20- trione 21-methanesulfonate (II). II in 18 ml. Me2CO was treated with a soln. of 250 mg. of NaI in 6 ml. Me2CO. This mixt. was refluxed with stirring for 15 min., cooled, and evapd. to dryness under reduced pressure to give 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.-hydroxy-21 -iodo-4- pregnene-3,11,20-trione. Similarly, II, gave 6.alpha.-methyl-9.alpha.-fluoro- 17.alpha.-hydroxy-21-iodo1,4-pregnadiene-3,11,20-trione (III). A suspension of 3.8 g. KF m a soln. of 7.7 g. 6.alpha.-methyl-9.alpha.-fluoro-11.beta.,17.alpha.,21-trihydroxy-4-pregnene-3,20-dione 21-methanesulfonate (IV) in 80 ml. Me2SO was stirred and heated on a steam bath 16 hrs. The mixt. was cooled, dild. with 500 ml. of HO, and extd. with CH2Cl2. The exts. gave a solid which was chromatographed on 200 g. of Florisil. The column was eluted with CH2Cl2 and mixts. of acetone (10 to 15%) in Skellysolve B. The material eluted with the acetone and Skellysolve B was extd. with acetone-Skellysolve B. The ext. was evapd. and the residue combined with the material eluted with CH2Cl2. This was chromatographed on Florisil and the column eluted with acetone (5 to 15%) in Skellysolve B. The material eluted with 10% acetone in Skellysolve B was recrystd. to give 0.48 g. 6.alpha.-methyl-9.alpha.-fluoro-11.beta.-hydroxy-17.alpha., 21-epoxy-4-pregnene-3,20-dione (V), m. 208-5.degree., v 3340, 1802, 16371600, 960, 870 cm.-1 Similarly a soln. of 6.alpha.-methyl-9.alpha., fluoro-17.alpha.,21-dihydroxy-4-pregnene-3,11,20-trione 21-toluenesulfonate in HCONMe2 heated with KF gave 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.,21-epoxy-4-pregnene-3,11,30-trione (VI). Similarly, 6.alpha.-methyl-9.alpha.-fluoro-11.beta., 17.alpha.,21-trihydroxy-1,4-pregnadiene-3,20-dione 21-methanesulfonate gave 6.alpha.-methyl-9.alpha.-fluoro-11.beta.-hydroxy-17.alpha.,21-epoxy-1,4-pregnadiene-3,20-dione (VII), m. 238-43.degree. [.alpha.] 150.degree. (acetone). Similarly, II gives 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.,21-epoxy-1,4-pregnadiene-3,11,20-trione (VIII). Other 6.alpha.- and 6.beta.- ***alkyl*** -9.alpha.- ***fluoro*** -11.beta., ***17*** .alpha.,21-trihydroxy- 1,4-pregnadiene-3,20-dione 21-sulfonates and their 11-oxo analogs react similarly to the corresponding 17.alpha.,21-epoxides. A mixt. of freshly prepd. Ag2CO3, 14 g. of 6.alpha.-methyl-9.alpha.-fluoro-11.beta., 17.alpha.-dihydroxy-21-iodo-4-pregnene-3,20-dione and 270 ml. MeCN were stirred and refluxed for 2 hours. The mixt. was cooled and filtered through Celite. The Celite was washed with hot MeCN; these washings were added to the filtrate. The dried filtrate was evapd. to dryness to give 12.3 g. of residue which was chromatographed on 900 g. of acid-washed alumina. The column was eluted with EtOAc (1 to 10%) in benzene. The material eluted with 10% acetate was recrystd. from acetone to give V. Similarly, 6.alpha.-methyl-9.alpha.-fluoro-11.beta.,17.alpha.-dihydroxy-21-iodo-1,4-pregnadiene-3,20-di-one yielded VII and III yielded VIII. V, VI, VII, VIII, and similar compds. have diuretic and antiinflammatory activity.

L22 ANSWER 13 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1962:448821 CAPLUS
DN 57:48821
OREF 57:9659h-i,9660a-c
TI Organic fluorine compounds. XXVI. The synthesis of fluorine compounds from sulfonic acid esters
AU Bergmann, Ernst D.; Shahak, I.
CS Hebrew Univ., Jerusalem
SO Bull. Res. Council Israel Sect. A (1961), 10, 91-9
DT Journal
LA Unavailable
AB cf. CA 56, 12728f. Alkyl sulfonates were prepd. from the corresponding alkoxides and PhSO2Cl or p-MeC6H4SO2Cl. A mixt. of 150 ml. diethylene glycol and 45 g. KF was dehydrated by heating at 100-10.degree. 30 min. with stirring. 0.5 mole of the alkyl sulfonate was added and the mixt. heated cautiously to 130-50.degree., depending on the b.p. of the fluoride formed, which was distd. over if boiling below 150.degree. or extd. with CHCl3. n-Pentyl, n-hexyl, n-heptyl, n-octyl, lauryl, and cetyl fluoride, 4-fluoromethyl-2,2-dimethyl-1,3-dioxolane (b. 133-4.degree.), 2-ethyl-4-fluoromethyl-2-methyl-1,3-dioxolane, Et .alpha.-fluoropropionate, and Et fluoroacetate were obtained in good yields (55-95%). Poorer yields (5-35%) were given by: 2-octyl fluoride, 1,3-dichloro-2-fluoropropane (b. 126.degree.): bis(.beta.-fluoroethyl) ether; 5-fluoro-1,3-dioxane (b. 103-5.degree.); 4-fluoromethyl-1,3-dioxolane (b. 111-12.degree.) 5-fluoro-2-phenyl-1,3-dioxane (b30 117-19.degree.); 4-fluoromethyl-2-phenyl-1,3-dioxolane (b30 111-14.degree.). In a second procedure, trioctylamine was neutralized with 40% HF and dried, giving probably C8H17NH2.HF, and fluorides were obtained from ***alkylsulfonates*** in ***17*** -52% yields. .alpha.- ***Fluoronitriles*** were obtained from aldehydes as follows: 0.6 mole of aldehyde, 25 g. NaCN (or 35 g. KCN) in aq. soln. treated with 0.5 mole PhSO2Cl in 120 ml. CH2Cl2 at 15.degree. with stirring for 1 hr. and at 25.degree. 6 hrs. Org. layer sepd., washed (H2O and aq. NaHSO3), dried, concd., and treated with KF gave fluoroacetonitrile (30%), .alpha.-fluoropropionitrile (b. 95-6.degree., 52%); .alpha.-fluorobutyronitrile (b. 115-16.degree., 58%); .alpha.-fluorovaleronitrile (b. 137-8.degree., 49%); .alpha.-fluoroisovaleronitrile (b. 126-7.degree., 60%) .alpha.-fluoroisocapronitrile (b. 132-3.degree., 57%). Alkyl-p-toluenesulfonates gave corresponding Cl, Br, I, CN, or CNS derivs. in similar procedures using LiCl, NaBr, NaI, KCN, or KCNS, in 72-96% yields.

L22 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1959:51316 CAPLUS
DN 53:51316
OREF 53:9297b-i,9298a-d
TI 6-Methyl-9.alpha.-fluoro-11-oxygenated pregnenes
IN Lincoln, Frank H., Jr.; Schneider, Wm. P.; Spero, Geo. B.
PA Upjohn Co.
DT Patent
LA Unavailable
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2867638		19590106	US	
DE 1058990			DE	
GB 863777			GB	

AB The prepn. of 6-hydrocarbyl substituted steroids was described. 6.alpha.-Methyl-9.alpha.-fluorohydrocortisone (1 g.) (cf. Spero, et al., C.A. 51, 11369h) and 10 ml. pyridine (I) cooled to 0.degree., 0.5 ml. Me2SO2Cl added, the soln. kept at 0-5.degree. 18 hrs., ice and 10 ml. H2O added, and then 120 ml. 5% HCl pptd. 6.alpha.-methyl-9.alpha.-fluoro-11.beta.,17.alpha.,21-trihydroxy-4-pregnene-3,20-dione 21-methanesulfonate (II), m. 166-78.degree. (decompn.). II (750 mg.) dissolved in 15 ml. acetone (III), refluxed with stirring 15 min. with 750 mg. NaI in 15 ml. III, and the soln. concd. to dryness in vacuo gave 6.alpha.-methyl-9.alpha.-fluoro-11.beta.,17.alpha.-dihydroxy-21-iodo-4-pregnene-3,20-dione (IV). IV stirred with 15 ml. HOAc 45 min., 750 mg. Zn dust added and stirring continued 20 min., the mixt. filtered, the filtrate dild. with methylene chloride and washed with aq. K2CO3 until neutral, the org. layer sepd., dried, concd. to 25 ml., and chromatographed over 40 g. Florisil gave 16 100 ml.-fractions (1 through 15 with Skellysolve B-hexane-10% III, and 16 with III). Fractions 5 through 16 combined and evapd. gave 80.3% 6.alpha.-methyl-9.alpha.-fluoro-11.beta.,17.alpha.-dihydroxy-4-pregnene-3,20-dione (V), m. 237-9.degree. (from III-Skellysolve B-hexanes), [.alpha.]D 103.degree. (in III). V (1.3 g.), 100 mg. chromic anhydride, 10 ml. glacial HOAc, and 0.5 ml. H2O kept 8 hrs. at room temp., poured into 50 ml. ice H2O, and neutralized with dil. NaOH pptd. 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.-hydroxy-4-pregnene-3,11,20-trione (VI) (crystd. from EtOAc and Skellysolve B-hexanes). In a similar manner to the prepn. of II, other 6.alpha.-alkyl(or .alpha.-aryl)-9.alpha.-fluorohydrocortisones or the corresponding fluorocortisones treated with the chlorides or bromides of toluenesulfonic acid, methanesulfonic acid, or other org. sulfonic acids gave the corresponding 21-substituted derivs. These compds. treated as in the prepn. of IV yielded the corresponding 21-iodo compds., such as 6.alpha.-ethyl-9.alpha.-fluoro-11.beta.,17.alpha.-dihydroxy-21-iodo-4-pregnene-3,20-dione (or the corresponding Pr, iso-Pr, Bu, iso-Bu, pentyl, Ph compds.), or 6.alpha.-methyl-9.alpha.-fluoro-17.alpha.-hydroxy-21-iodo-4-pregnene-3,11,20-trione (or the corresponding Et, Pr, iso-Pr, Bu, iso-Bu, pentyl, hexyl compds.). Treating the diones or triones thus obtained by the process used in the prepn. of V (or with Na bisulfite or thiosulfate) yields the corresponding 6.alpha.- ***alkyl*** -9.alpha.- ***fluoro*** -11.beta., ***17*** .alpha.-dihydroxy-4-pregnene-3,20-diones or 6.alpha.- ***alkyl*** -9.alpha.- ***fluoro*** - ***17*** .alpha.-hydroxy-4-pregnene-3,11,20-triones. Instead of the 6.alpha.-methyl-9.alpha.-fluorohydrocortisone (or cortisone) the 6.beta.-epimer can be used in this series of reactions. If the reaction conditions are kept near neutrality, the corresponding 6.beta.-epimers are formed, which yield the 6.alpha.-epimers on treatment with acid or base in an org. solvent. 6.alpha.-Methyl-11.beta.,17.alpha.-dihydroxy-4-pregnene-3,20-dione (1 g.), 650 mg. N-bromoacetamide, and 6 ml. I stirred in the dark 30 min., cooled in ice, a stream of SO2 directed onto the surface of the stirred mixt. until a neg. KI-starch test was obtained, 50 ml. H2O added, and the mixt. maintained at 5.degree. 30 min. pptd. 0.75 g. 6.alpha.-methyl-17.alpha.-hydroxy-4,9(11)-pregnadiene-3,20-dione (VII). VII (0.5 g.) dissolved in 20 ml. methylene chloride, 1 ml. 71% perchloric acid in 10 ml. H2O and 200 mg. N-bromoacetamide in 50 ml. tert-BuOH added, the mixt. maintained at room temp. 15 min., mixed with 0.3 g. Na sulfite in 12 ml. H2O, the resulting mixt. distd. in vacuo until the residual soln. became cloudy, and 100 ml. ice H2O added pptd. 6.alpha.-methyl-9.alpha.-bromo-11.beta.,17.alpha.-dihydroxy-4-pregnene-3,20-dione (VIII) (crystd. from III-Skellysolve B-hexane). VIII (0.45 g.), 0.45 g. anhyd. KOAc, and 20 ml. III refluxed 5 hrs., cooled, poured into H2O, extd. with methylene chloride, the ext. dried, chromatographed on 25 g. Florisil, and the column developed with Skellysolve B-hexane contg. increasing amts. of III yielded 6.alpha.-methyl-9(11)-oxido-17.alpha.-hydroxy-4-pregnene-3,20-dione (IX) in the eluate contg. 10% III. IX (1 g.) in 50 ml. methylene chloride, 5 ml. 48% HF, and 0.5 ml. 71% perchloric acid stirred 6 hrs. and poured into excess cold aq. 5% NaHCO3, the org. layer dried, and chromatographed as in the prepn. of IX gave V. Other 6.alpha.-alkyl(or aryl)-11.beta.,17.alpha.-dihydroxy-4-pregnene-3,20-diones or the 6.beta.-epimers will react similarly to give the corresponding 9.alpha.fluoro derivs. (X). X oxidized with chromic anhydride as in the prepn. of VI yielded 6.alpha.-alkyl(or aryl)-9.alpha.-fluoro-17.alpha.-hydroxy-4-pregnene-3,11,20-triones; the 6.beta.-epimers react similarly. V (2 g.), 1 g. 3-oxobisnor-4-cholen-22-

al, and 115 ml. dimethylformamide added to a growing culture of Septomyxa affinis (ATCC 6737) and incubated 24 hrs., the steroidal material extd. from the filtrate with methylene chloride, the ext. evapd. to dryness, chromatographed on a Florisil column, and the column developed with 5 fractions each of methylene chloride, Skellysolve B-III mixtures (9: 1, 8:2, 7:3, 1:1), and MeOH gave 6.alpha.-methyl-9.alpha.-fluoro-11.beta.,17.alpha.-dihydroxy-1,4-pregnadiene-3,20-dione (recrystd. from III, m. 292-303.degree.) from the 7:3 Skellysolve B-III fraction. V and VI (and their 6.beta.-epimers) cause a loss of salt and water and also possess antiinflammatory, glucocorticoid, uterine, ovarial, and adrenal growth-depressional and adrenocorticoid activity. They are used in oral, parenteral, and topical compns.; in the latter case, incorporation of an antibiotic in the ointment has therapeutic advantages.

L22 ANSWER 15 OF 15 CAPLUS COPYRIGHT 2002 ACS
AN 1959:17487 CAPLUS
DN 53:17487
OREF 53:3298c-i
TI 6-Fluoro-17.alpha.-alkynyl-4-androstenes
IN Herr, Milton E.; Babcock, John C.; Campbell, J. Allan; Hogg, John A.; Pederson, Raymond L.
PA Upjohn Co.
DT Patent
LA Unavailable
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2838549		19580610	US	
GB 893149			GB	

AB The prepn. of 6-fluoro-11.beta.,17.beta.-dihydroxy-17.alpha.-ethynyl-4-androsten-3-one (I), 6-fluoro-11.beta.,17.beta.-dihydroxy-17.alpha.-ethynyl-19-nor-4-androsten-3-one (II), 6-fluoro-17.beta.-hydroxy-17.alpha.-ethynyl-4-androstene-3,11-dione (III), 6-fluoro-17.beta.-hydroxy-17.alpha.-ethynyl-19-nor-4-androstene-3,11-dione (IV), the 17-acylates, and intermediates is described. 6-Fluoro-11.beta.-hydroxy-3-(N-pyrrolidinyl)-3,5-androstandien-17-one (2.25 g.) in 80 ml. anhyd. tetrahydrofuran added to a liquid NH3 soln. contg. CH.tplbond.CK, the mixt. kept in the cold several hrs., dild. with NH4Cl soln., extd. with Et2O, concd., the residue refluxed several hrs. in a buffered soln. of 6 g. NaOAc, 6 ml. H2O, 4 ml. AcOH, and 40 ml. MeOH, the mixt. extd. with Et2O, washed, evapd., and the residue in CH2Cl2 chromatographed on Florisil gave the 6.alpha.-isomer of I. The 6.beta.-form was present in the mother liquors and could be sepd. by addnl. chromatography followed by crystn. Similarly, substituting K alkylacetylides was productive of the corresponding 6- ***fluoro*** -11.beta.-hydroxy- ***17*** .alpha.-(***alkylethynyl***)testosterone. The 6.alpha.-form of I (1 g.) refluxed with (EtCO)2O yielded 6.alpha.-fluoro-11.beta.,17.beta.-dihydroxy-17.alpha.-ethynyl-4-androsten-3-one 17-propionate (V). Similarly the 6.beta.-form of I gave the 6.beta.-isomer of V. Similarly, 6-fluoro-11.beta.-hydroxy-3-(N-pyrrolidinyl)-19-nor-3,5-androstadien-3-one with KC.tplbond.CH in NH3, treated with aq. NH4Cl, and the resulting enamine hydrolyzed with NaOAc, AcOH, H2O, and MeOH gave II. The substitution of K alkylacetylides gives the corresponding 17.alpha.-alkylethynyl derivs. In the same manner as described above II may be converted to its 17-propionate or other 17-acylates. I (0.425 g.) in 20 ml. AcOH left several hrs. at 25.degree. with 0.25 g. CrO3, 1 ml. H2O, and 20 ml. AcOH gave 6.alpha.-form of III. In like manner the 6.beta.-form of III was prepd. Similarly other 17.alpha.-alkylethynyl derivs. of I are oxidized to give the corresponding analogs of III. III (6.alpha.-form, 1 g.) with (EtCO)2O gave the 17.alpha.-propionate (VI). The 6.beta.-form of VI is similarly prepd. from the 6.beta.-form of III. In the same manner shown above other 17-acylates of III are prepd. Also the 17-propionates of the 17.alpha.-alkylethynyl analogs of III are similarly prepd. II (6.alpha.-form) oxidized as above with CrO3 gave IV (6.alpha.-form). The 6.beta.-form of IV was similarly obtained by oxidation of the 6.beta.-form of II. Similarly the 17.alpha.-methylethynyl and other 17.alpha.-alkylethynyl analogs of II were oxidized to give the corresponding 17.alpha.-alkylethynyl analogs of IV. IV (6.alpha.- or 6.beta.-form) may be converted to the 17-propionate or to other 17-acylates. The 17-propionates of 6- ***fluoro*** -17.beta.-hydroxy- ***17*** .alpha.- ***alkylethynyl*** -19-nor-4-androstane-3,11-diones may be similarly obtained.

=> d his

(FILE 'HOME' ENTERED AT 11:18:52 ON 06 JUN 2002)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 11:19:22 ON 06 JUN 2002
L1 2613 S FERTILITY (3A) TREAT?
L2 78848 S CONTRACEPT?
L3 81300 S L1 OR L2
L4 142 S ENDOMETRIUM (3A) MATUR?
L5 81423 S L3 OR L4
L6 19 S L4 AND L3
L7 29173 S PROGEST? (3A) RECEPTOR?
L8 1077 S (ANTAGONIST OR INHIBIT?) (3A) L7
L9 0 S 17 ALPHA FLUORALKYL?
L10 0 S 17 FLUORALKY?
L11 8 S 17 (3A) FLURO?
L12 3684 S 17 (3A) FLUOR?
L13 15 S L12 (3A) ALKYL?
L14 0 S L13 AND L8
L15 69 S L8 AND L3
L16 1 S L15 AND L6
L17 33312 S CONCEPTION?
L18 35752 S L17 OR L1
L19 0 S L18 AND L4 AND L8
L20 1 S L8 AND L4
L21 8 DUP REM L6 (11 DUPLICATES REMOVED)
L22 15 DUP REM L13 (0 DUPLICATES REMOVED)

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		120.87	121.08

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) TOTAL	ENTRY	SINCE FILE SESSION	
CA SUBSCRIBER PRICE		-11.77	-11.77

STN INTERNATIONAL LOGOFF AT 11:44:55 ON 06 JUN 2002